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THE
LARYNGOSCOPE.

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NEW INVESTIGATIONS ON THE PHYSIOLOGY OF THE LABYRINTHINE FLUIDS.*†

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Three years ago we reported before this Society the results of preliminary experiments in rabbits designed to investigate some questions pertaining to the physiology of the labyrinthine fluids. Using the method employed by Weed in his studies of the resorption of the cerebrospinal fluid, small amounts of solutions of iron-ammonium citrate and potassium ferrocyanide were introduced into the subarachnoid space. The animals were killed 15 to 120 minutes after the injection and the temporal bones fixed in formalin-hydrochloric acid solutions. Since the iron salts form precipitates of Prussian blue granules with the hydrochloric acid, the iron solutions could be traced through this reaction into various structures of the labyrinth. From their distribution certain conclusions were drawn regarding the existence of currents within the labyrinthine fluids and possible places of resorption of endo- and perilymph.

This paper reports the results of further experiments in rabbits, cats and monkeys.

*Read at the Fifty-fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif., May 27, 1950.

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EXPERIMENTS ON RABBITS.

In 15 rabbits, after withdrawal of some spinal fluid, 0.2 to 1 cc. of a 1 per cent or 5 per cent iron-salt solution was injected into the cerebellomedullar cisterna. In view of the possibility of a rapid resorption of the salts within the cochlea, the animals were killed one to 15 minutes after the injection.

After injection of the 1 per cent solution, the salts could be detected after six minutes in the cochlea in only one rabbit out of five, and this to a very limited degree; after injection of the 5 per cent solution, the salts were found in the cochlea in five rabbits out of seven. The most important factor in the extent and speed of penetration seems, therefore, the concentration of the solution.

The distribution of the granules within the various structures of the cochlea indicates a typical pattern for the spread of the salts. Once they have entered the scala tympani they can be traced upward to the helicotrema and from there downward into the scala vestibuli.

Only a part of the salts which enter the scala tympani. however, passes upward towards the helicotrema; another portion goes laterally into the stroma of the lower part of the spiral ligament and from there into the small venous spaces within the latter. Still another portion goes through the basilar membrane into the cochlear duct and into the various spaces between the cells of the organ of Corti. From there, the salts can be traced either in a lateral direction into the spiral ligament, mostly through the lower parts of the prominentia spiralis and the sulcus spiralis externus, or medially into the tissue and the small venous spaces of the crista spiralis or through the perineural spaces of the cochlear fibres into the spiral ganglion. From the spiral ligament precipitations of the salts can be followed, sometimes within, but mostly along the somewhat larger vessels through the interscalar septa into the modiolus. From the crista spiralis they can be traced in the veins into the modiolus. Nearly all the salts which have entered the scala vestibuli through the heli-

cotrema penetrate either through Reissner's membrane into the cochlear duct or into the uppermost part of the spiral ligament or into the crista spiralis. In 15 minutes only a very small part of the salts reaches the perilymphatic spaces of the vestibule. Here they can be traced into the lumen of the trabecular vessels. All the salts which have entered the cochlear duct within the same period of time are resorbed there and none have passed through the reunient duct into the saccule or beyond. In five rabbits granules were found in the medial (inner) wall of the endolymphatic sac. Their presence is most probably the result of an intravital penetration from the subarachnoid space through a circumscribed area of the adjacent dural wall along a projection of the dura.

In those cases in which only smaller amounts of the salts have entered the scala tympani, they are resorbed either into the spiral ligament on the lateral wall alone or through the basilar membrane into the cochlear duct of the first and successive portions of the second turn. The findings indicate that the resorptive mechanism within the cochlea is highly effective and able to cope with even relatively large amounts of iron salts. From the fundus of the internal auditory meatus the salts extend along the perineural spaces of the cochlear nerve into the spiral ganglion of the basal turn and into the stroma of the maculae and cristae. This penetration is, however, never extensive and always insignificant in comparison with the penetration through the cochlear aqueduct.

These findings in the rabbits confirm to a large extent the observations reported in our preliminary report. An important new finding seems the demonstration of a spread of the salts from the subarachnoid space through a circumscribed area of the dura into the tissue of the endolymphatic sac and from there into some of the blood vessels. Granules found in this area, or even in the lumen of the sac, are not, therefore, as previously assumed, conclusive evidence of a current directed from the cochlea into the sac. The penetration of granules from the perilymphatic spaces into the lumen of some of the trabecular vessels deserves great interest because it indicates resorption of the perilymph in these areas. Fur-

thermore, it was demonstrated that the spread of the salts through the interscalar septa into the modiolus occurs primarily not within the blood vessels but in the perivascular spaces along the vessels.

EXPERIMENTS ON CATS.

In a second series of experiments 0.1 cc. of a 1 per cent solution was injected in four cats directly into the scala tympani through the round window membrane after exposure and opening of the bulla tympanica. The injections were made on both sides, but a few minutes apart. The animals were killed five to 16 minutes after the injection.

In each of the cats a far larger amount of the solution entered the scala tympani than in any of the rabbits. In this way a heavy overloading of the resorptive mechanisms of the cochlea was effected. A part of the iron salts injected into the scala tympani went into the aqueductus cochleae and emerged from there into the subarachnoid space. This "reverse flow" is a manifestation of a simple diffusion of the salts or the result of an increased pressure within the scala during the injection which forces the fluid in this direction.

The major portion of the injected fluid, however, spreads within the cochlea in the same directions as in rabbits injected intrathecally. Although the amounts of iron salts which reach the perilymphatic cisterna of the vestibule are larger than in rabbits, they are still comparatively small, a fact which again demonstrates the effectiveness of the resorptive mechanism of the cochlea. Similarly, only a small amount of the salts which have passed from the scalae into the cochlear duct is not resorbed from the duct, and enters the saccule through the reunient duct.

The salts which have penetrated into the perilymphatic spaces of the vestibule from the scala vestibuli might spread from there into the perilymphatic spaces of the semicircular canals. They might enter the lumen of some of the small veins in the trabecula of the perilymphatic tissue or penetrate, mainly along the perivascular spaces, through the inner wall

of the vestibule into the arachnoid spaces of the fundus of the internal auditory meatus. Within the perilymphatic spaces themselves larger accumulations of salts were found, particularly on the outer surface of the walls of the maculae of the saccule and utricle and of the cristae ampullares, and also around the nerves which run from the vestibular or ampullar wall to these structures. These accumulations indicate the presence of movements of the salts to these areas. The extension of granules from these locations along the nerve fibres into the stroma of the maculae, and to a lesser extent also of the cristae and also in the opposite direction, along the nerve fibres and sometimes in the lumen of small vessels towards Scarpa's ganglion and the arachnoid meshwork of the fundus, indicates a subsequent spread into the opposite directions. In some instances the salts have evidently passed from the stroma of the maculae and cristae into the lumen of the endolymphatic spaces. There is also some evidence of a penetration of the salts through other parts of the walls of the membranous labyrinth into the lumen.

The salts which entered the saccule could be traced from there through the saccular duct into the endolymphatic duct and sac, but in none of these cases was a larger number of granules found in these cavities and in none was there evidence of resorption of the salts from the sac into the perisaccular tissue within 16 minutes after injection into the scala tympani. The precipitated granules in the lumen of the utricle and the semicircular canals seem to have come exclusively from the perilymphatic spaces by way of penetration along the nerve fibres at the indicated areas or directly through the membranous walls.

EXPERIMENTS ON MONKEYS.

In a third series of experiments nine Rhesus monkeys were studied.

In one instance only nine minutes after the intrathecal injection granules were found within the cochlear aqueducts and within the labyrinthine cavities. Within the cochlea they

showed approximately the same distribution as in the rabbits and the cats. Other granules could be traced from the fundus of the internal auditory meatus along the perineural spaces of the saccular and vestibular nerves into the respective maculae. There were no granules in the lumen of the lowest part of the cochlear duct, the saccule or the saccular duct. Numerous granules were found, however, in the tissue spaces and the small blood vessels of the perisaccular tissue surrounding the rugose portion of the endolymphatic sac becoming less numerous in the distal part of the sac. The granules decreased in intensity towards the lumen, but some were found there, sometimes within the phagocytes. It would be hardly conceivable that within nine minutes all these granules should be carried through an endolymphatic current all the way down from the cochlear duct without leaving any traces in the intervening parts. Penetration of the solution from the subarachnoid space through a circumscribed area of the dura into the perisaccular tissue and into the blood vessels, and to a limited extent into the lumen, similar to the movement of the salts described in the rabbits, seems more probable.

The greater length and the narrowness of the aqueductus cochleae in monkeys seem mainly responsible for the fact that in only one case out of nine the granules had penetrated into the cochlea.

DISCUSSION.

The spread of the injected iron salts in the rabbits and monkeys from the cerebellomedullar cisterna into the fundus of the internal auditory meatus and the cochlear aqueduct could be the result of the currents within the cerebrospinal fluid together with the action of all the physicochemical factors governing the distribution of dissolved substances within fluids. Biological membranes encountered by the dissolved iron salts could modify but not materially change the spread towards the inner ear. The assumption of a special fluid current directed from the subarachnoid space towards the inner ear seems unnecessary. Such a fluid current has, however, been postulated because certain insoluble substances, such as

India ink or animal charcoal, introduced into the subarachnoid space might also penetrate into the inner ear. Sometimes in man, blood is found in the scala tympani following a hemorrhage into the subarachnoid space. A spread of these substances into the aqueductus cochleae, which is in a certain sense only a recess of the subarachnoid space, cannot be taken, however, as convincing proof for the existence of such a fluid current. Since the particles in question are very small, their spread into all parts of the subarachnoid space, their penetration into the scala tympani, and their passage from the fundus of the internal auditory meatus into the modiolus or other parts of the inner ear could again be the result of the physicochemical factors governing the distribution of small particles.

Although the possibility that a certain small proportion of the perilymph originates from the cerebrospinal fluid and enters via the cochlear aqueduct and perhaps also through the internal auditory meatus cannot be definitely denied, the cerebrospinal fluid does not seem an essential source of the perilymph. Most probably there is only very little, if any, actual fluid exchange between the subarachnoid space and the labyrinthine cavities.

The distribution of the iron salts within the various structures of the cochlea cannot be explained solely by the action of physicochemical factors. The accumulation of granules in certain areas of the spiral ligament and in the crista spiralis and the relative freedom from granules of other areas, such as the stria vascularis, indicates the action of specific biological factors. It points to a resorption of the iron salts in certain areas into the tissue spaces. From there the salts can be traced into blood vessels or into fluid spaces which surround the modiolar vessels and from the latter into the fundus of the internal auditory meatus. The main resorption of the iron salts seems to take place from the cochlear duct, and only to a lesser extent directly through the scalae. This is facilitated by the free permeability of the basilar membrane and Reissner's membrane for the salts. Since salts are only resorbed in dissolved form together with fluids, fluid move-

ments must exist, directed in the scalae towards the lower and upper parts of the spiral ligament and in the cochlear duct towards the external spiral sulcus, the spiral ligament and the crista spiralis. The spread of the salts within the scala tympani upward to the helicotrema and from there downward through the scala vestibuli is, therefore, at least partly controlled by the actual fluid movements within the scalae and by the fact that a large-scale penetration through the basilar membrane and Reissner's membrane takes place. The biological forces behind these fluid movements are similar to those controlling the production and resorption of the cerebrospinal fluid.

A partial confirmation of our views is furnished by the histological findings of Saxen according to whom desquamated and degenerated epithelial cells and other debris in the cochlear duct in dogs and man are removed through the resorptive action of the epithelium of the sulcus spiralis externus. The extent to which endolymphatic fluid is also removed in the same way could not be found out by the histologic methods employed by Saxen. That this resorption of fluid actually takes place is proved, however, by our experiments.

The conclusion arrived at by us, that a very active resorption takes place in the cochlear duct, does not contradict the existence of a fluid current within the cochlear duct directed towards the endolymphatic duct and sac as indicated by the experiments of Guild.

The results of our experiments, however, seem to demonstrate quite clearly that the current from the cochlea to the endolymphatic sac is without great significance for the resorption of low molecular substances in physiological concentrations from the cochlea. The latter will be disposed of by the resorptive mechanism within the cochlear duct. Only when excessive amounts penetrate into the cochlear duct, as in Guild's guinea pigs or in our cats, will they be carried down to the sac. The metabolites resulting from the function of the cells of the organ of Corti which penetrate into the endolymph are evidently also removed by this local resorptive mechanism. It would be hard to understand why, for instance,

metabolites originating in the apical turn of the cochlea should be carried all the way down through the cochlear duct into the saccule and thereby come in contact with other parts of the organ of Corti.

The resorption from the cochlear duct of high molecular substances, of emulsions of fats, and of corpuscular elements is perhaps less efficient than that of the low molescular substances used in our experiments. It is conceivable that larger proportions of them might reach the endolymphatic sac.

That portion of the salts which was not resorbed from the scalae eventually reaches the vestibule, particularly when their concentration in the perilymph was as great as in our cats injected through the round window. In these instances, the iron salts had also spread from the scala tympani through the cochlear aqueduct into the subarachnoid space. This would have been difficult to explain if a strong fluid current from the subarachnoid space into the scala tympani had really existed as assumed by many authors; however, it becomes easily understandable if one assumes that there is no fluid current, or hardly any, within the cochlear aqueduct, and that salts can spread through the latter in either direction, depending upon their relative concentration in the cerebrospinal fluid and in the labyrinthine fluids.

The fate of the salts which have gotten into the vestibule has already been discussed in the cat experiments.

The spread from the vestibule to the fundus of the internal auditory meatus gives rise to the same questions as the spread from the scala tympani to the subarachnoid space. It would be incompatible with the existence of a strong fluid current from the fundus into the labyrinth, and one comes to the conclusion again that such a current does not exist and that a spread might occur in either direction, depending upon the relative concentration of the salts in the fundus and the vestibule.

Nothing definite can be said about the main source of the perilymphatic fluid. It seems much more probable that it is

derived from the perilymphatic blood vessels than that it originates from the endolymph and passes through the walls of the membranous labyrinth. The walls of the membranous labyrinth seem to be easily penetrated by low-molecular substances, and this could explain why the peri- and endolymph are virtually isosmotic. The fact that in our experiments the iron salts moved from the peri- into the endolymphatic space simply demonstrates the high degree of permeability of the membranes and should not be taken as an indication that this is the prevailing direction of movement of dissolved substances under physiological conditions. Normally it is more likely to be from the endolymph towards the perilymph.

The resorption of the cochlear perilymph seems to take place from both scalae into the tissue spaces and from there into the blood vessels of the spiral ligament, from the vestibule and the semicircular canals into the blood vessels of the trabecula of the perilymphatic tissue. The spread into the perivascular and perineural spaces is probably of lesser importance under physiological conditions. Both spaces are more likely to represent auxiliary avenues of escape for dissolved substances if their concentration is so high that the normal pathways have become overloaded.

The salts which have passed into the lumen of the saccule can be traced from there into the endolymphatic duct and sac. Because resorption of endolymph takes place in certain areas of the endolymphatic sac, the salts have probably been carried down to these areas not solely by diffusion but also by an actual fluid current. That no evidence of resorption from the lumen of the sac was seen in any of our specimens could have been due to the relatively short time interval between the injection of the salts and the death of the animals.

Great caution is advisable in the interpretation of the findings in and around the endolymphatic sac because of diffusion of the salts from a circumscribed area of the subarachnoid space along a projection of the dura into the perisaccular tissue. Here the presence of granules could easily be mistaken as evidence of resorption from the lumen or of transudation

from the blood stream. There was nothing in the distribution of the granules in our specimens which could be interpreted as an indication of a current from the utricle to the endolymphatic sac.

The possibility of a local resorption of endolymph from the lumen of the saccule and utricle into the stroma of the maculae and from the ampullae into the stroma of the cristae must be considered, although the histologic findings in the examined cases do not permit any definite conclusions regarding these questions.

SUMMARY AND CONCLUSIONS.

Using the method of Weed, iron salt solutions of various concentrations were injected into the subarachnoid space of 15 rabbits and nine Rhesus monkeys. Similarly, in four cats iron salts were injected through the round window membrane directly into the scala tympani.

The iron salts spread readily through the wide aqueductus cochleae of the rabbits and less readily through the narrow aqueduct of the monkeys into the scala tympani. Another less extensive spread may occur from the fundus of the internal auditory meatus mainly in the perineural spaces of the respective nerves into the modiolus, the maculae and the cristae ampullares. Membranes encountered by the iron salts do not seem to slow up this spread perceptibly. In the cats the iron salts spread in the opposite direction; from the scala tympani through the aqueduct into the subarachnoid space, from the modiolus, the vestibule and the perilymphatic spaces of the semicircular canals into the fundus of the internal auditory meatus.

These findings are regarded as an indication that active fluid currents are absent in either direction, and that the spread of the iron salts is mainly or entirely the result of the action of the usual physicochemical factors governing the distribution of dissolved substances within fluids.

In the scala tympani the salts spread partly upward to the helicotrema and partly through the basilar membrane into the

cochlear duct, while another part was resorbed into the spiral ligament. From the helicotrema the salts spread in the scala vestibuli towards the vestibule. A part of them passed through Reissner's membrane into the cochlear duct, and another part was resorbed directly into the spiral ligament. If sufficient amounts of iron salts reach the vestibule, they spread from there into the perilymphatic spaces of the semicircular canals and eventually into the blood vessels of the trabecula of the perilymphatic tissue. Other portions pass through the walls of the membranous labyrinth into the endolymphatic space. If the concentration of the salts in the perilymph is very high, they may spread along the perineural and perivascular spaces in either direction towards the maculae and cristae and into the fundus of the internal auditory meatus. The findings indicate that actual resorption of perilymphatic fluid takes place mainly in three areas: from both scalae into the spiral ligament and from the vestibule and the semicircular canals into the blood vessels in the trabecula of the perilymphatic tissue.

Since no perceptible fluid current towards the perilymphatic space could be demonstrated, neither from the subarachnoid nor from the endolymphatic space, at least the main part of the perilymphatic fluid is evidently formed in the perilymphatic space itself, most likely from the blood vessels. The marked permeability of the walls of the endolymphatic system makes it probable that a spread of dissolved substances takes place from the endo- into the perilymphatic fluid or vice versa.

Within the cochlear duct resorption of iron salts occurs through the sulcus spiralis externus and the spiral ligament, and through the crista spiralis, indicating the resorption of endolymph in the same areas. The resorptive mechanism of the cochlea seems more than sufficient for the disposal of low molecular substances in physiological concentrations.

Resorption of the endolymph from a certain part of the endolymphatic sac, although not visible in the specimens, seems an established fact and indicates the presence of an evidently slow fluid current towards the sac. This current seems to affect mainly those substances which are difficult for

resorption by the regular cochlear mechanism and are carried into the sac. Presence of granules in the subepithelial tissue of the endolymphatic sac is, however, not necessarily an evidence of resorption from the lumen because of diffusion of the salts from a circumscribed area of the subarachnoid space along a projection of the dura into the perisaccular tissue.

MODERN TEMPORAL BONE SURGERY — HISTORY OF ITS EVOLUTION.*

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Thirty years ago when I began to practice otology I was not quite satisfied with the results that I personally obtained in temporal bone surgery, and on observing the results of others I was not more satisfied with theirs. Especially disconcerting to me at that time were the extremely poor results obtained with the traditionally accepted and employed surgical treatment of obstructive exostosis of the external auditory canal.

When exostosis of the external auditory canal becomes sufficiently obstructive to interfere with visualization of the tympanic membrane and to interfere with transmission of sound to the middle ear, or with drainage of an acute or chronic middle ear infection, surgical removal of the bony mass becomes essential, whatever its size, extent or location may be.

The accepted and most commonly employed surgical procedure for many years for the cure of such a condition consisted of enlarging the meatus through a postauricular incision, by removing the posterior bony wall of the external auditory canal and making a conchal flap, as in the postauricular radical mastoidectomy, and lining the mastoid cavity with skin grafts. The operation involved too much trauma and too much unnecessary destruction of healthy tissue for the removal of an innocent mass of bone from within the external auditory canal. The consequences in many of these cases were compli-

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†Lempert Institute of Otology.

‡Lempert Research Foundation, Inc.

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cations, sometimes intracranial. It all seemed very unnecessary to me, and I decided to look for and find a way of surgically removing an exostosis lesion from the external auditory canal without opening and destroying the mastoid process.

That decision marked the beginning of my endaural surgery. I operated upon my next case of exostosis as follows: I made an incision along the anterior margin of the concha down to the bony mastoid cortex, then subperiosteally elevated the skin and periosteum covering the exostosis mass with a narrow periosteal elevator slowly, a few millimeters at a time. This elevation was done slowly because the obstructive bony mass is as a rule irregular in shape and contour, and if an attempt is made to separate the cutaneous lining, which becomes thinner and thinner as it approaches the hidden tympanic membrane, it may be injured and torn; therefore, I separated a few millimeters at a time. With a small electrically driven polishing burr I then removed as much of the exostosis mass from the posterior, inferoposterior and superoposterior walls as was exposed to view. This having been done, I continued my elevation of the skin and periosteum a few millimeters at a time and gradually removed the exostosis mass wherever found down to the anterior mastoid cortex without opening any of the mastoid cells until the tympanic ring was reached. After removing the entire bony mass I replaced the skin and periosteum into its normal position against the anterior mastoid cortex. The entire circumference of the tympanic membrane became visible and the operation was over.

I then packed the canal with paraffin mesh gauze, removing it at the end of 48 hours. Incision is sutured. Primary union is effected. This is the complete procedure. The many men who have since tried this procedure of mine would never again think of opening a mastoid process to remove an exostosis from the external auditory canal. I have employed this method ever since and found it to be the most satisfactory procedure for the otologist and for the patient. It can be done under local or general anesthesia and hospitalization need not be more than one or two days (see Figs. 1-5).

ENDAURAL REMOVAL OF OBSTRUCTIVE EXOSTOSIS FROM EXTERNAL AUDITORY CANAL.

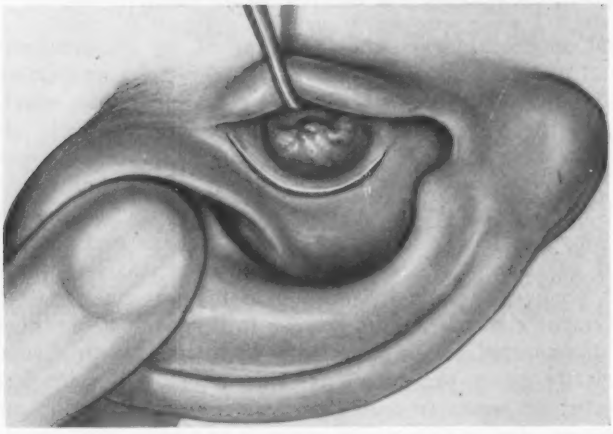

Fig. 1. Step I. Endaural incision along anterior margin of concha.

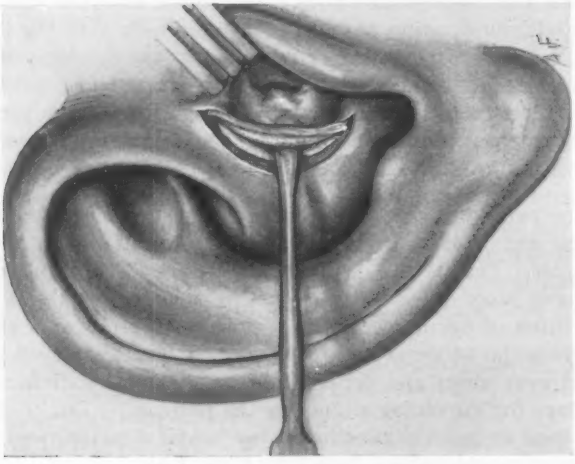

Fig. 2. Step II. Subperiosteal elevation of skin and periosteum from region of exostosis mass.

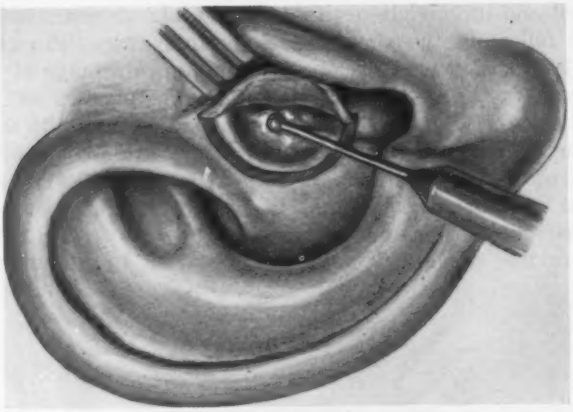

Fig. 3. Step III. Exostosis mass is removed with electrically driven burr.

Fig. 4. Step IV. Exostosis mass was removed.

POSTAURICULAR FISTULA.

One of the commonly met conditions in those days and which confronts us occasionally today, calling for surgical correction by the otologist, was the permanently open post-

auricular fistula. Such a fistula was either *1.* the result of arrested and incomplete healing of the postauricular mastoid wound following the simple mastoidectomy when the postauricular incision was left open to maintain drainage of the infection; *2.* the repeated infection and breakdown of the healed postauricular scar following the simple mastoidectomy because of repeated recurrence of acute attacks of otitis media, or *3.* the sloughing and breakdown of the freshly sutured postauricular mastoid incision following the postauricular radical mastoidectomy.

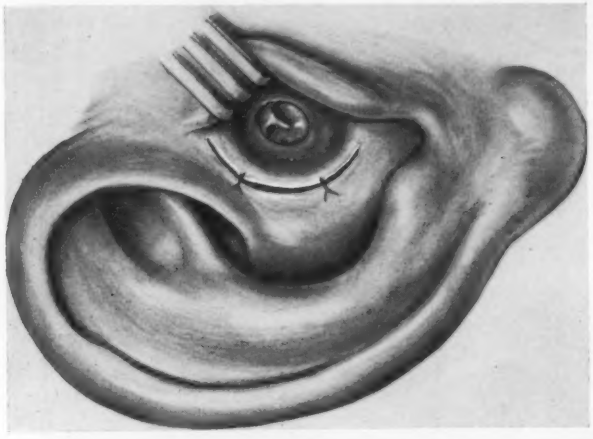

Fig. 5. Step V. Normal contour of external auditory canal has been re-established. The tympanic membrane is now visible. The subperiosteally elevated skin and periosteum is replaced and held in position with paraffin mesh gauze.

Very many methods of surgically closing such a postauricular fistula were variously employed by otologists. The underlying surgical principle of all these methods was the borrowing and diverting of skin, periosteum and also muscle from the neighborhood of the postauricular fistula for the correction and closure of the fistular defect. The results obtained following these surgical efforts were not satisfactory as a rule and I thought that there was something wrong and that there should be a means of permanently closing such a fistula without sacrificing and deforming the healthy tissue around it. After observing that a postauricular fistula is

always either completely or partially lined with a funnel-shaped invagination of skin and periosteum, I decided to proceed as follows, again endaurally:

I made an endaural incision along the anterior margin of the concha, down to the mastoid cortex through the skin and periosteum, and with a narrow periosteal elevator, I separated and elevated the auricle and postauricular skin and periosteum subperiosteally in a posterior direction up to the margin of the outer and wider circumference of the funnel-shaped invagination of skin and periosteum lining the postauricular fistula. The intention being to mobilize the auricle so that I could freely move it in any direction I wished. By slowly and gently elevating the inwardly adherent skin and periosteum lining the fistula through the eudaural incision with the narrow periosteal elevator, I everted outward the funnel of skin and periosteum. By converting the inwardly directed funnel of skin and periosteum into an outwardly directed funnel without severing it from its continuity with the postauricular skin and periosteum I was confronted with more skin and periosteum than needed for closing the postauricular fistula. If there are any infected cells — if an incomplete mastoidectomy had been done and there is any infection within that region — I remove the pathologic processes. With fine scissors I removed the excessive skin and periosteum and then sutured the freshly cut edges of skin and periosteum. I put a little rubber tissue drain into the original incision made endaurally, which was removed at the end of five days. A perfectly endaural and postaural wound was the result. This can be done under local or general anesthesia. The procedure is being carried out with the greatest of satisfaction by many men all over the world today (see Figs. 6-12).

COMPLETE ENDAURAL SUBCORTICAL MASTOIDOTYMPANECTOMY.

The most commonly encountered postauricular fistulas in those days were secondary to the performance of the postauricular radical mastoidectomy, and these were the most difficult to correct. While correcting these fistulas I could not

LEMPERT PLASTIC FOR CLOSURE OF POSTAURICULAR FISTULA.

Fig. 6. Postauricular fistula.

help thinking, "Why do we get fistulas following the radical mastoidectomy?" "Couldn't a means of performing a radical

Fig. 7. Step I. Endaural incision through skin and periosteum is made along anterior margin of concha.

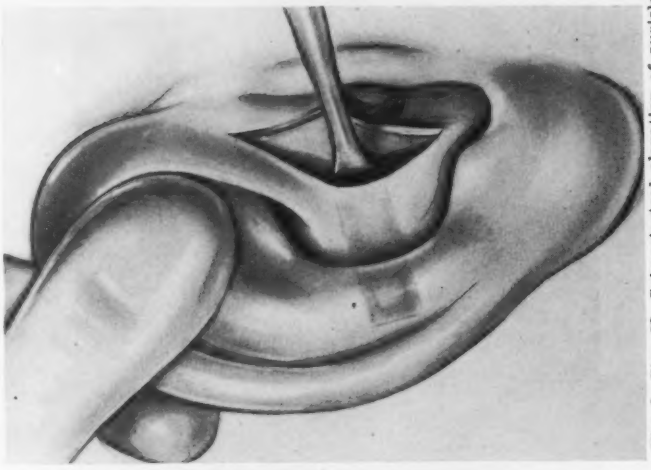

Fig. 8. Step II. Subperiosteal elevation of auricle from its bony attachments through endaural incision.

mastoidectomy be devised which would render the occurrence of a postauricular fistula impossible?"

On analyzing the whys and wherefores of fistula following the radical mastoidectomy it seemed natural that a fistula should occur when one considered the technique of performing a postauricular radical mastoidectomy. First of all we make a postauricular incision in order to open the mastoid antrum and to exenterate the pathology from the mastoid process. Can we do a radical mastoidectomy which necessitates also the removal of the pathologic processes from within the tympanic cavity, through a postauricular incision? We cannot, and that is the reason why we must continue to destroy and remove the posterior bony wall of the external auditory canal plus the skin and periosteum covering it, in order to expose to view the tympanic cavity and render it accessible for surgical intervention. In other words, when doing a radical mastoidectomy postauricularly, we cannot reach the tympanic cavity surgically without destroying also the posterior wall of the external auditory canal; we cannot

Fig. 9. Step III. Subperiosteal elevation and eversion outward of inverted skin lining postauricular fistula through the endaural incision. The postauricular skin deficiency is thus converted into a skin surplus.

reach it through the postauricular approach unless we supplement it with an endaural approach. To speak of the Schwartze radical mastoidectomy as a postauricular technique is really a misrepresentation of the facts, since in reality it is a combined postauricular and endaural technique. It became quite obvious to me that a technique which involves such wholesale destruction of soft tissue surrounding the auricle will undermine and weaken the whole structural foundation of the auricle, and as a result it is quite natural in the presence of infection for the sutured postauricular incision to become infected and to open up with a resulting permanent postauricular fistula.

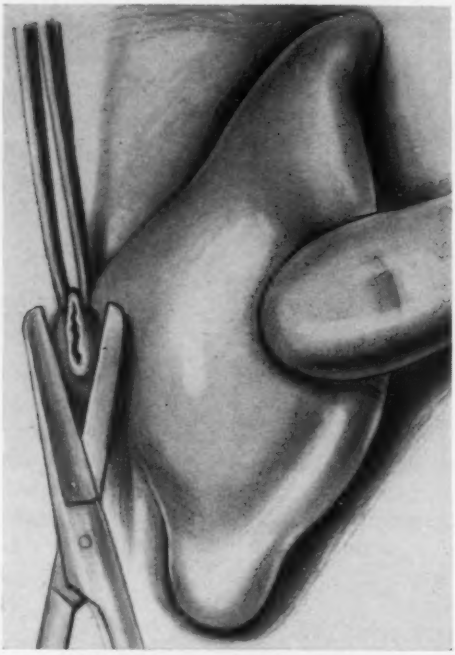

Fig. 10. Step IV. Removal of everted skin surplus.

It thus became quite obvious to me at the time that if I could do a radical mastoidectomy without a postauricular incision, the occurrence of a postauricular fistula would no longer be possible. I decided to try to do the radical mastoidectomy endaurally only and without a postauricular incision. After trying various endaural incisions I finally concluded that the best endaural approach for a radical mastoidectomy requires the following four endaural incisions: The first incision is made along the posterior margin of the tympanic ring through skin and periosteum; the second one is made along the entire superoposterior wall of the external auditory canal coming right out of the canal up to the apex of the supra-

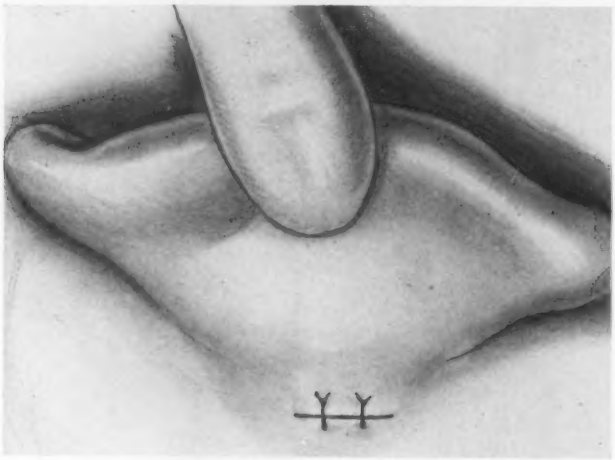

Fig. 11. Step V. Closure of postauricular fistula by suturing the edges of skin and periosteum.

Fig. 12. Step VI. The antauricular incision is sutured and postoperative mastoid wound drained. External auditory canal is packed with paraffin mesh gauze.

meatal membranous triangle; the third incision is begun at the inferior margin of the first incision and is carried outward along the inferoposterior wall towards the lower end of the anterior margin of the concha. The fourth incision follows the anterior margin of the concha up to the apex of the suprameatal triangle and connects the second and third incisions. The skin and periosteum is then elevated from its bony attachments, removed and conserved for skin-grafting the postoperative cavity.

I have since performed all my mastoidotympanectomies endaurally and, of course, the occurrence of postoperative postauricular fistulas following this surgery has become a thing of the past.

While doing my radicals endaurally, without ever again getting postauricular fistulas as a result, and constantly observing critically my results with this surgery, I asked myself what was it that the patient, for whom one would advise a radical mastoidectomy, or what I prefer to call a mastoidotympanectomy, expected of the surgeon as a result of submitting to this surgical procedure. The patient says, "I have had a running ear for 10 years (or perhaps 20 or 30 years). There is a bad odor to the discharge. I have had every form of treatment. I have been to many doctors. Can you do something to stop this discharge?"

Following a thorough examination the otologist would tell the patient that what he needs is a radical mastoidectomy. The reply of the intelligent patient would be, "That is fine, but will it stop my discharge?" The young otologist with little experience, fearing that he will not succeed in removing all the pathology responsible for the middle ear discharge, usually replied, "I cannot promise you that the operation will stop your discharge, but it will protect you from intracranial complications." His uncertainty of succeeding to remove all the pathologic processes encountered was based upon his fear of injuring one or more of the many vital anatomic structures within the temporal bone by his blind search for the mastoid antrum in a small sclerotic mastoid process through the outer

mastoid cortex, or by attempting to remove the pathology covering and hiding from view the vital anatomy within the tympanic spaces. His constant fear of injuring the vital anatomy was due to the fact that in performing the Schwartze postauricular radical mastoidectomy he had no other safeguard except the hope of avoiding injury to the anatomy which most often he could not see. These same mental reservations which prompted him not to promise the patient a dry ear should also have prevented his suggesting that the possible onset of an intracranial complication could thus be prevented. Even the extremely skillful otologist, after extensive experience with the postauricular radical mastoidectomy technique, will not infrequently encounter difficulties beyond his expectation. Being fully cognizant of the inherent difficulties and shortcomings of the Schwartze postauricular radical mastoidectomy technique, I have developed a simple and safe mastoidotympanectomy technique. Any relatively young in experience, well-trained otologist can employ this technique without fear of injuring vital anatomy and with full confidence in his ability to remove all the pathologic processes responsible for the persistent middle ear discharge. The electric headlight and the magnifying loupe are invaluable assets to the safe performance of this technique.

TECHNIQUE.

The endaural subcortical mastoidotympanectomy technique is based upon the following surgical principles:

1. The abandonment of the hazardous and tedious blind search for the mastoid antrum through the outer mastoid cortex. Too many accidents occur unnecessarily as a direct result of this search.

2. The exploration of all the vital anatomy and eradication of the pathologic processes within the confines of the mastoid portion of the temporal bone by first removing the thinner anterior wall of the mastoid cortex with the electrically driven burr directed from within outward, beginning with the tympanic margin of the posterior bony wall of the external

auditory canal, instead of directing the instrumentation blindly and dodgingly, first through the much thicker outer wall of the mastoid cortex towards the vital anatomy.

3. The exploration of the pathologic process within the epitympanum by engaging the tympanic margin of the superior bony wall of the external auditory canal with the electrically driven burr and removing this bony wall from within outward until the roof of the external auditory canal and the roof of the epitympanum become continuous.

4. The exploration of the pathologic process within the hypotympanum by engaging the tympanic margin of the inferior bony wall of the external auditory canal with the electrically driven burr and removing this bony wall from within outward until the floor of the hypotympanum and the floor of the external auditory canal become continuous.

5. The removal of the pathologic processes from the tympanum, epitympanum and hypotympanum which rest upon the thickened diseased mucoperiosteum lining these tympanic spaces, by elevating, separating and stripping the entire mucoperiosteum from its attachment to the tympanic bony walls and removing it together with the pathologic processes resting upon it. All the vital middle ear anatomy is thus exposed to view without predisposing it to the danger of injury. This method of removing the pathology from the middle ear spaces is analogous to the removal of the damaged rug or carpet with the dirt on it, instead of removing the dirt from the already damaged carpet.

If one hopes to obtain a dry ear as a result of this surgery, one must remove all the pathological processes encountered, any one of which when not removed could perpetuate the middle ear discharge. The removal of granulations, polypi and cholesteatomata from the mastoid process, epitympanum, tympanum and hypotympanum is not sufficient to stop a chronic middle ear discharge. For very many years there existed an infection within the middle ear, the mucoperiosteum lining, it is infected, thickened and inflamed, and one has to remove it until one exposes and inspects the bony

structure underneath it. The perieustachian tube cells which are infected must be removed, but a curette must never be introduced into the Eustachian tube. The use of a curette in

LEMPERT SUBCORTICAL MASTOIDOTYMPANECTOMY.

Fig. 13. Four endaural incisions are made through skin and periosteum.

the mouth of the Eustachian tube as a rule results in osteomyelitis of the bony portion of the Eustachian tube. With a No. 11 or 12 S.S. White dental excavator the granulations around the stapedial crura must be removed, and one must also remove the granulations from the round window niche,

not only to remove a source of continued infection but also in the hope of giving the patient better hearing than he enjoyed before the operation. A necrotic facial canal must be removed

Fig. 14. Skin flap removed and saved for skin grafting postoperative bony cavity.

and the facial nerve exposed to view. A necrotic bony carotid canal must be removed and carotid artery exposed to view. Finally, if it is desired to have a dry ear the tensor tympani muscle lying within its semicanal must be removed. This

muscle has been bathing in pus for very many years and a continued postoperative middle ear discharge may well result when this infected muscle is not removed. This must be done by fracturing the processus cochleariformis in a direction towards and over the facial canal, then stripping the muscle

Fig. 15. The outer mastoid cortex, spina henle and the entire anterior mastoid cortex can now be seen.

from its semicanal towards the Eustachian tube orifice and finally grasping it with a narrow pointed artery clamp and pulling it out *in toto*. The chances of obtaining a dry ear with

the above described technique are almost 100 per cent providing the after-treatment is carried out with the same undivided attention (see Figs. 13-25).

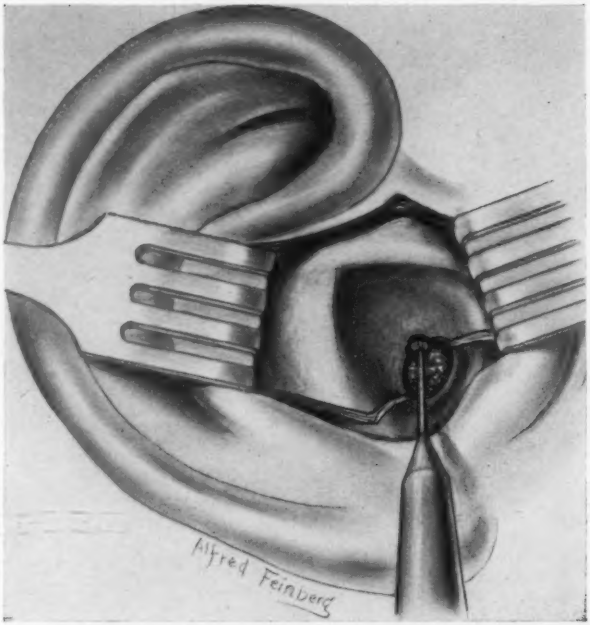

Fig. 16. The tympanic inner margin of the posterior wall of the external auditory canal is engaged with an electrically driven sharp cutting burr and the anterior wall of the mastoid cortex is gradually removed from within outward.

FACIAL NERVE INJURY REPAIR.

When temporal bone surgery is performed with proper lighting and magnification it can be done without danger of injuring the vital anatomy involved. I do not mean to say

that one may not occasionally injure the facial nerve. That can happen, but when it happens we must know that it happened and how and when it happened. In those circumstances

Fig. 17. The incudo stapedial joint and the external semicircular canal can be seen as result of partial removal of the anterior mastoid cortex from within outward.

we have the "right" to have an accident. But when we have an accidental injury of the facial nerve while doing a radical mastoidectomy and do not know it until the anesthetist tells us. "The face is crooked"; and we with surprise respond by

saying "How did that ever happen? — I wasn't even near the facial nerve." Well, this is very sad, because the surgeon is the only one who should have known how it ever happened.

Fig. 18. Following the removal of the entire anterior wall of the mastoid cortex from within outward and the exenteration of the diseased mastoid cell structure the sharply defined semicircular canals can now be seen.

One cannot overemphasize the fact that if there is an accident we must know how it happened, where it happened, and when it happened; and if we do not know, we have no right to be doing temporal bone surgery because such a confession in the

face of an obviously existing injury is concrete evidence that we never knew where we were in the temporal bone. Accidents can happen to anybody, but if I am doing this operation

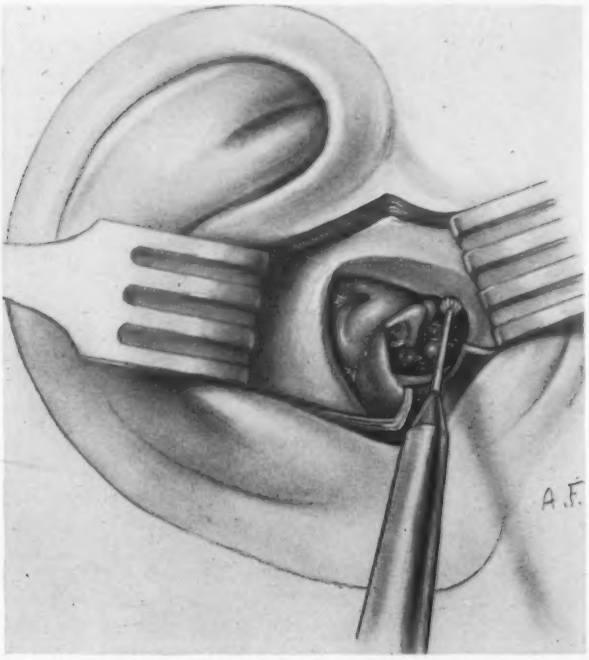

Fig. 19. To expose the pathologic process within the epitympanum the innermost margin of the superior bony wall of the external auditory canal is removed from within outward to establish a continuity between the roof of the canal and the floor of the middle fossa.

properly it is I who will tell the anesthetist, long before he tells me, "Will you look at that face? I have just injured the facial nerve." The anesthetist's response will inevitably be: "Yes! The face is crooked." The anesthetist having thus con-

firmed the fact that I have injured the facial nerve, my only comment to his answer is, "Thank you."

Fig. 20. To expose the pathologic process within the hypotympanum the tympanic margin of the bony floor of the external auditory canal is engaged with an electrically driven burr and removed from within outward until a continuity between the floor of the external auditory canal and the floor of the hypotympanum is established.

The most common facial nerve injury is the result of an inward fracture of bone into the Fallopian canal which compresses the facial nerve. This can be readily observed with magnifying glasses. It takes less than one minute to remove

such a bony fragment. After removing it, the facial nerve is further decompressed by removing a few millimeters of the Fallopian canal, both anterior and posterior to the fractured area. As a result of such immediate attention the facial

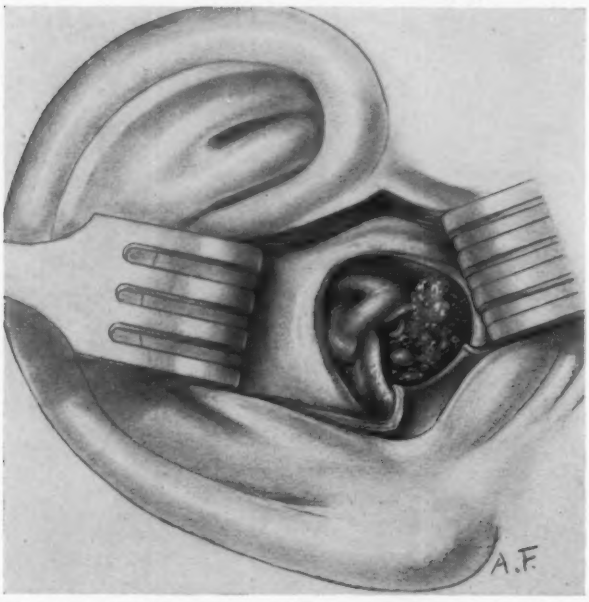

Fig. 21. The pathologic processes within the tympanum, epitympanum and hypotympanum are seen now exposed to view and resting upon the thickened and diseased mucoperiosteum.

deformity disappears immediately on the operating table, and our troubles are over. When the facial nerve has accidentally been cross-sectioned without creating a gap between the sectioned ends, the immediate uncapping of the Fallopian canal

for a distance of about 5 mm. in either direction of the injury will as a rule result in healing of the nerve injury with ultimate restoration of good function; however, when the facial nerve has been torn asunder with a resulting gap between the

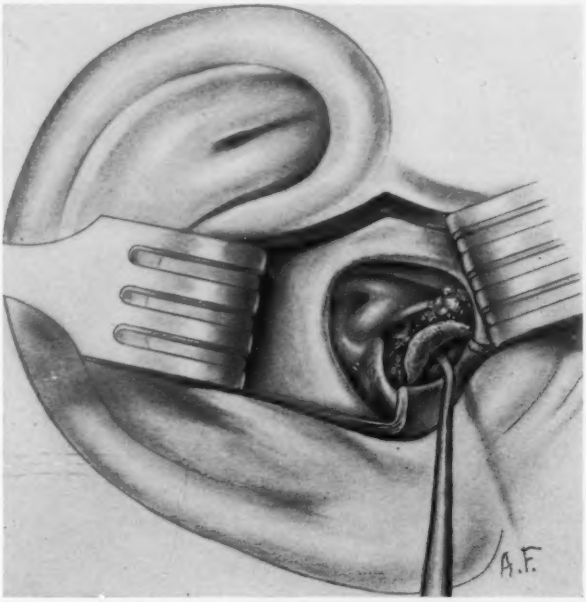

Fig. 22. The diseased mucoperiosteum together with the pathologic processes resting upon it are being removed by separating and elevating the mucoperiosteum from its attachment to the bony tympanic walls.

severed ends I found that by changing the normal habitat of the nerve I could establish a favorable status for quick repair by employing the end to end anastomosis technique.

Since a straight line is the shortest distance between two points, I displace the facial nerve from its curved canal and convert the curved line of the nerve into a straight line. Immediately, no matter how much nerve has been missing as a result of the injury, we have much more nerve tissue left

Fig. 23. The pathologic processes resting upon the mucoperiosteum having been removed, the infected tensor tympani muscle within its semicanal is now exposed to view.

than is needed to meet end to end. After trimming both torn ends to where both meet comfortably but not too loosely to form a straight line, they are held in apposition with a fine suture.

End to end suture of the injured facial nerve has given me better end-results than I was able to obtain with the Ballance-Duell transplantation technique. The end to end anastomosis technique is a much simpler one than the transplantation

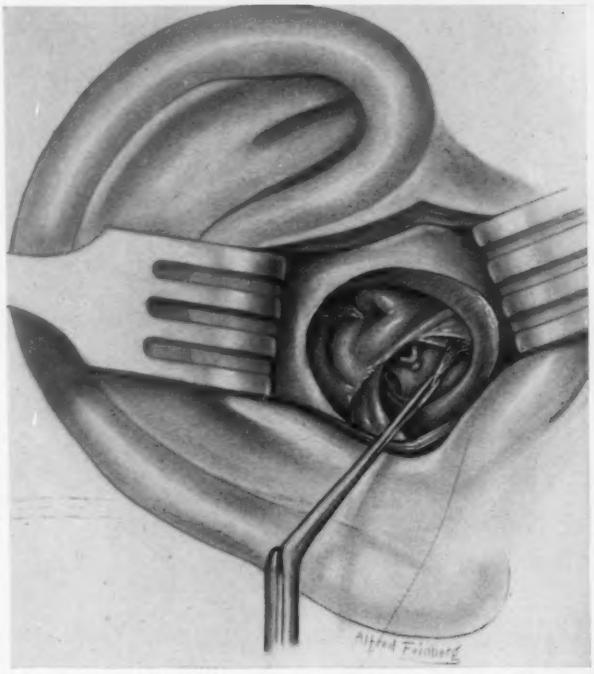

Fig. 24. The infected tensor tympani muscle is removed from its semicanal.

technique. Using your magnifying glasses and proper lighting, you are the master of the situation. When I say "master of the situation" I repeat that any otologist doing temporal bone surgery must necessarily be master of the situation. He

must be able to repair the facial nerve injury immediately on the table. He must know where, when and how he has caused any injury. The general surgeon, if he meets with an accident on the table, does not call for help and does not procrastinate; he does not permit the patient to leave the operating table,

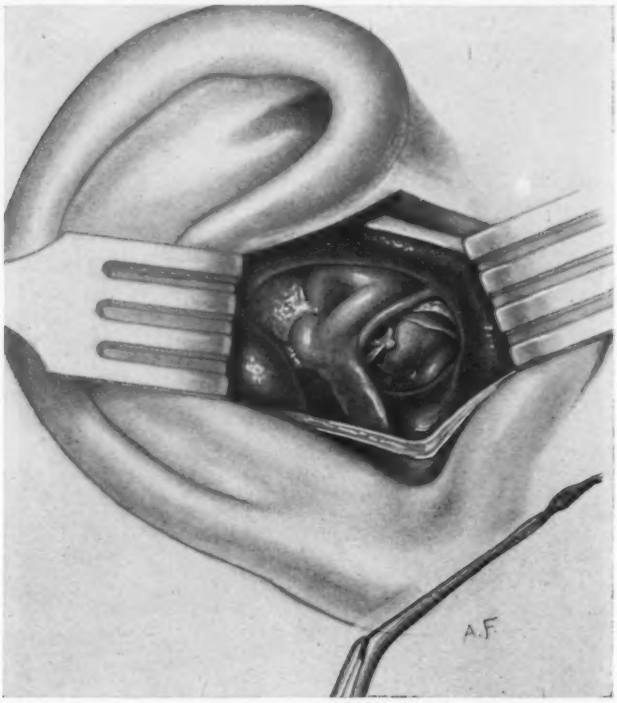

Fig. 25. The outer wall of the mastoid cortex finally removed, exposing to view all the vital anatomy within the completely exenterated mastoido-tympanic wound.

and he certainly would never think of discharging the patient from the hospital without personally repairing the injury in question, or having someone of greater skill repair it for him. He is, as a rule, the master of the situation; he immediately handles whatever accident he meets. Otologists must be trained to do the same (see Figs. 26-29).

LEMPERT END TO END ANASTOMOSIS OF INJURED FACIAL NERVE.

Fig. 26. The most commonly encountered facial nerve injury as a result of temporal bone surgery is the inward fracture of the bony wall of the facial canal.

COMPLETE ENDAURAL MASTOIDECTOMY.

Years ago when performing the simple mastoidectomy for acute suppurative mastoiditis in children, one of the problems which frequently haunted us was the child who was repeatedly and periodically brought back to the otologist with a ballooned-out, inflamed postauricular scar long after the postauricular mastoid wound had completely healed. This was usually the result of a newly contracted middle ear infection from varying causes. One customarily told the mother that it was just a middle ear infection, a very simple thing with secondary inflammation of the postauricular scar requiring

Fig. 27. Severed facial nerve is seen.

incision and drainage, and that was all. Should that patient, for some reason, have gone to one's colleague, even one's best friend, it was too often a different story. Then this inflamed ear was labeled as a recurrent mastoiditis, creating indirectly the impression that the original mastoidectomy had been incompletely performed. As a matter of fact, it has been my observation that the more completely a mastoidectomy was performed the more likely was this clinical picture to appear subsequent to a postoperatively occurring otitis media. The so-called recurrence happened only because it was the natural result. When we have a middle ear infection in the presence of a normal healthy mastoid process which was never operated upon, the course of least resistance for the infection within the middle ear is the tympanic membrane, and either

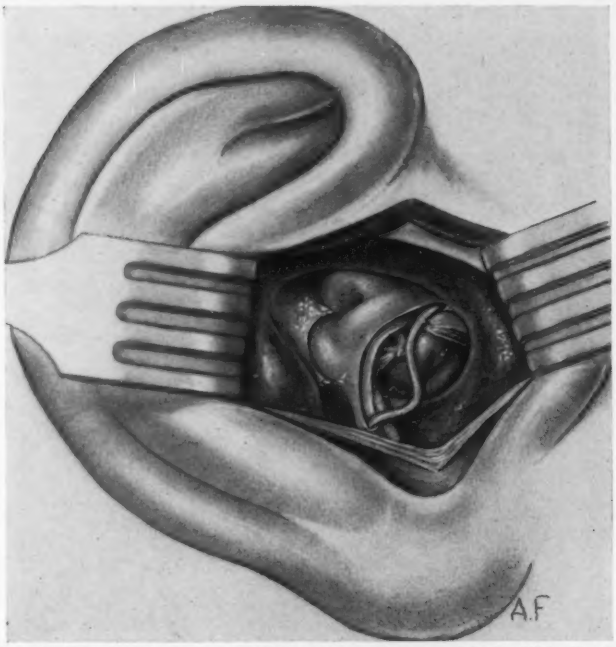

Fig. 28. The facial nerve is dislodged from its curved bed within the bony facial canal.

the membrane ruptures by itself or, if seen in time by the otologist, he incises it, drains it, and hopes for the best. When a child has had a previous mastoidectomy which thoroughly healed and then contracts a middle ear infection, the course of least resistance for such a middle ear infection is no longer the tympanic membrane. With that mastoid antrum widely open and the mastoid process widely exenterated, the infection gradually again takes the course of least resistance, which this time, however, is backward to the mastoid wound instead of forward to the tympanic membrane; and the postauricular scar tissue whose resistance is much lower than that of a healthy postauricular skin and periosteum becomes infected immediately and "balloons out." In other words, infection naturally always takes the course of least resistance.

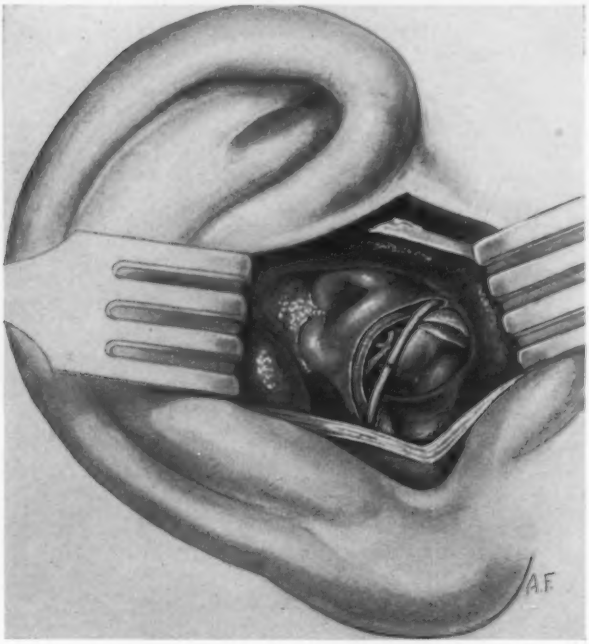

Fig. 29. Since a straight line is the shortest distance between two points there is no need for nerve transplantation. End to end suture is employed.

Based on that observation, I felt a little tired of having all these children brought back to me every six months, and I determined to do something about it. It occurred to me that if I were to perform my simple mastoidectomies endaurally it should make it impossible for this to happen, because they would not have the postauricular scar to become inflamed secondary to a recurrent middle ear infection. A normal healthy postauricular skin and periosteum would offer more resistance to a postoperatively occurring middle ear infection than the tympanic membrane, and this membrane would instead become inflamed, bulge and rupture as it did before the original mastoidectomy was performed.

LEMPERT ENDAURAL MASTOIDECTOMY.

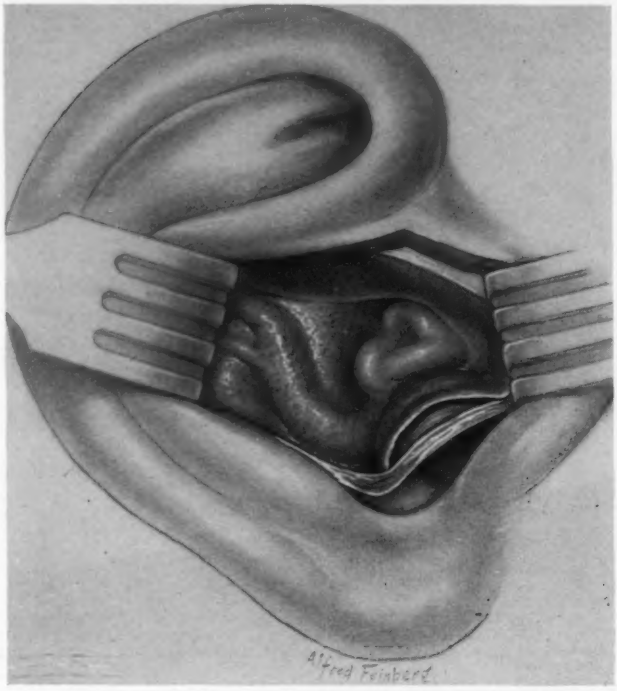

Fig. 30. Completely exenterated mastoid process is seen.

TECHNIQUE.

I have been performing my mastoidectomies for acute suppurative mastoidtis through the endaural approach for more than 20 years. The outline of the anterior cartilaginous margin of the concha underneath the cutaneous cover is brought into prominence by placing the index and middle fingers of the left hand tightly against the face of the concha and pulling and stretching the concha backward against and over the outer mastoid cortex. With the point of the knife directed backward towards the outer mastoid cortex, instead of inward and parallel with the course of the posterior canal wall, the

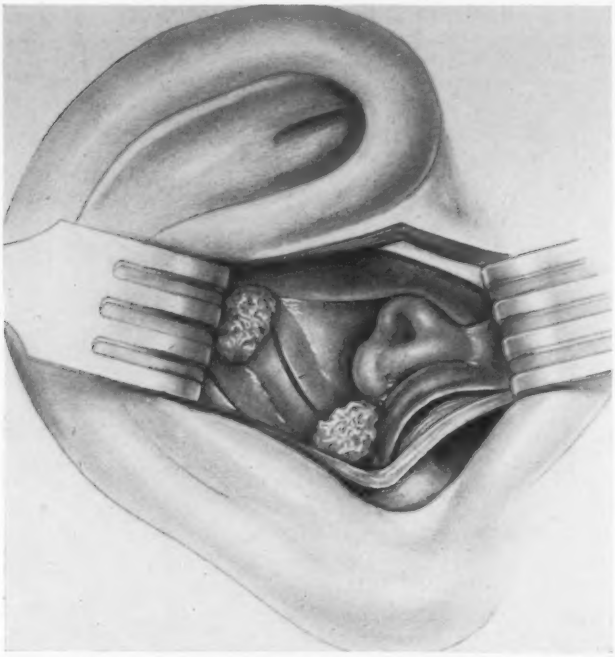

Fig. 31. The lateral sinus is tamponaded at its entrance to and exit from the mastoid process. Lateral sinus wall is incised and thrombus removed from its lumen.

antauricular endaural incision through skin and periosteum is carried immediately adjacent to the entire anterior margin of the concha and upward along the anterior aspect of the helix up to the apex of the suprameatal membranous triangle. When this endaural incision is made correctly an exposure of the mastoid process is obtainable, after subperiosteal elevation of the skin and periosteum from the outer cortex and insertion of the retractors, which is more adequate for surgical intervention than the exposure obtainable as a result of a postauricular incision.

Through this endaural approach I have always without difficulty surgically treated the intracranial complications such

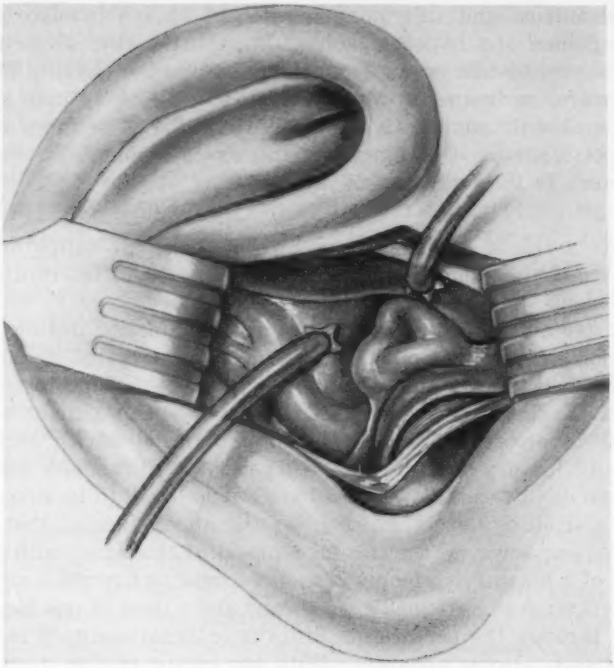

Fig. 32. Middle and posterior fossa abscess is drained with rubber tubing.

as sinus thrombosis, temporosphenoidal abscess, and cerebellar abscess when encountered, and without supplementing the postauricular incision. Today, when the fenestration operation is being performed endaurally by otologists everywhere, the possibility and desirability of doing all temporal bone surgery through the endaural approach is no longer questioned (see Figs. 30-32).

ENDAURAL MASTOIDOATTICOTOMY (MODIFIED RADICAL).

One of the frequently encountered clinical otologic entities requiring surgical attention is the one caused by a chronically persisting and progressive suppurative lesion within the mas-

toid antrum and epitympanum without having involved the tympanum and hypotympanum. Though the clinical picture presented by this type of otologic lesion and the physical findings are too frequently much more innocent looking than that of a chronic suppurative lesion within the tympanum and hypotympanum, the danger of intracranial complications is greater in the former than in the latter.

As a rule a patient suffering from a chronic suppurative epitympanic lesion gives a history of periodic intermittent purulent middle ear discharge for a number of years which resisted all medical therapy. Physical examination during the period of active middle ear discharge will reveal a fairly normal looking tympanic membrane plus a small perforation in the region of Shrapnell's membrane. If such a patient happens to present himself for examination and advice during one of his dry periods, the perforation in Shrapnell's membrane can be easily overlooked since it is likely to be covered by a small dry crust which gives the appearance of that of cerumen; however, the careful removal of this crust with the aid of a magnifying loop will soon uncover and reveal a small perforation in Shrapnell's membrane and a drop of pus escaping through the perforation into the external auditory canal will soon become apparent. With the escape of this drop of pus tiny shreds of cholesteatoma will become visible within the confines of the epitympanum. In the well advanced case of epitympanic suppuration the patient may give a history of one or more recent attacks of vertigo. In the presence of such a history the slightest pressure exerted upon the region of the perforation with a cotton applicator will evoke severe nystagmus and vertigo. Fistulas of the vestibular labyrinth which rarely accompany chronic suppurative lesions of the tympanum and hypotympanum in the presence of a large central perforation of the tympanic membrane commonly occur in chronic suppurative lesions of the epitympanum with small perforations in Shrapnell's membrane. This is due to the prolonged pressure exerted in the region of the external semicircular canal by the gradually increasing cholesteatomatous mass which surrounds the incudomalleolar joint. The hearing

loss for airborne sound accompanying epitympanic lesions is as a rule insignificant and rarely descends to a level lower than 30 db for the three speech frequencies, whereas the hearing loss accompanying chronic suppurative lesions involving the tympanum and epitympanum is as a rule much greater. That surgical eradication of the pathologic process is the only available means of permanently curing this type of chronic suppurative otitis media was well recognized by otologists for very many years; however, methods employed for eradicating the pathology in this type of case were either too radical or inadequate. Even the most adequate and conservative methods employed years ago were in many instances inconsiderate of the future welfare of the ear involved. Many otologists were too radical in their surgical intervention, because they would invariably perform a radical mastoidectomy in such cases with no consideration for the further loss of hearing which must result from such surgery.

Other otologists in their desire to conserve the still existing fairly good hearing would perform what they called a more extensive mastoidectomy and widened the communication between the aditus and antrum, by removing the zygomatic cells. Such surgical intervention was inadequate because it did not provide the necessary accessibility for the removal of the cholesteatomatous mass surrounding the incudomalleolar joint which wound itself around it like cotton threads around a spool. Some well trained and experienced otologists would perform a thoroughly adequate mostoidoatticotomy and successfully remove all the pathology, but they also removed the incus. Though the removal of the incus in such cases need not always result in a further loss of hearing, such removal, however, prevents the occasional improvement in hearing acuity which follows the removal of the pathology which partially immobilized the ossicular chain; furthermore, in the presence of a fistula in the vestibular labyrinth, which is not an infrequent finding in this type of chronic suppurative otitis media, the mastoidoatticotomy technique employed in those days did not provide means for sealing and protecting the membranous

labyrinth in the fistula region against degenerating with subsequent total loss of hearing or from becoming infected in the distant future, with subsequent intracranial extension of the infection. Though the immediate welfare of the ear involved was provided for, its distant future welfare was not protected.

Based upon the above mentioned observations made with the surgical treatment of the chronic suppurative lesions within the epitympanum, I developed and employed for the last 14 years the following endaural mastoidoatticotomy technique:

TECHNIQUE.

In perfecting the mastoidoatticotomy technique I considered it essential that the following three objectives be reached as a result of this surgery:

1. The removal of the pathologic process from the epitympanum must be accomplished without disturbing the ossicular chain or injuring the tympanic membrane, so that the preoperative hearing acuity could be conserved. Postoperative further loss in hearing must be avoided.

2. The postoperative mastoid wound must be permanently segregated and excommunicated from the middle ear to prevent the spread of any subsequent middle ear infection to it and its involvement by it.

3. In the presence of a labyrinthine fistula, which is a common finding in such cases, a means of permanently sealing such a fistula to assure the survival of the membranous labyrinth must be provided so that the still existing hearing function may be conserved.

After performing a complete endaural mastoidectomy and exenterating all the pathologic processes encountered within the mastoid process, the aditus is widened, and the mastoid aspect of the posterior, superoposterior and superior bony walls of the external auditory canal is skeletonized. This results in the exposure to view of the incudomalleolar joint

and the pathologic processes surrounding it within the epitympanum. The posterior, superoposterior and superior bony walls of the external auditory canal are removed from the skin and periosteum adherent to it, without either lacerating the cutaneous lining of the canal or severing it from its attachment to the tympanic membrane. The pathologic processes encountered within the epitympanum are carefully removed without disturbing the ossicular chain or injuring the tympanic membrane. A viable cutaneous pedicle flap is plastically fashioned from the skin and periosteum from which the bony walls of the external auditory canal have been previously removed, taking care not to sever the cutaneous flap from its attachment to the tympanic membrane. The tym-

LEMPERT ENDAURAL MASTOIDOATTICOTOMY.

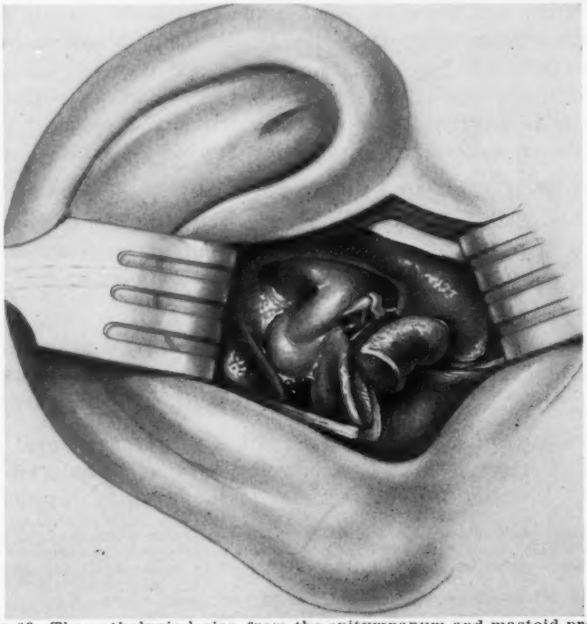

Fig. 33. The pathologic lesion from the epitympanum and mastoid process was removed. The viable tympanomeatal pedicle flap for sealing the tympanic space and isolating and excommunicating the mastoid process from the tympanic space is seen.

panomeatal flap thus created is then carefully trimmed and so placed in the mastoid wound that it will seal off and isolate the epitympanum, the tympanum and hypotympanum from communicating with the mastoid process and will also seal a labyrinthine fistula when present (see Fig. 33).

CONCLUSION.

The viable tympanomeatal pedicle flap which I have created as part of the endaural mastoidoatticotomy technique for the surgical treatment of chronic epitympanic suppuration, suggested and made possible my subsequent creation and development for the first time in the history of otology the now universally employed, practical one-stage fenestration operation for the restoration of practical hearing in clinical otosclerosis.

SUPRAHYOID NECK INFECTIONS.*†‡

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Suprahyoid infections differ from other neck infections in certain clinical features. It seems, however, that all infections of the neck share in the confusion of fascial spaces and in the confusion of terminology.

It must be emphasized that the fascial spaces are clinical as well as anatomic. They have become clinical entities because *disease* has invaded them so frequently and has produced such characteristic signs and symptoms. It has been *disease*, rather than regional anatomy, which has given these areas their clinical importance. For example, the peritonsillar space would have attracted little attention had not *disease* dissected between the tonsillar capsule and the pharyngeal aponeurosis. Likewise, the pharyngomaxillary space would have received scant publicity had not pharyngeal *disease* extended laterally beyond the superior constrictor muscle.

With anatomy remaining essentially the same, terminology is important in allowing a common ground for discussion. It becomes necessary, therefore, to state in advance of a clinical report the anatomic area involved and the terminology to be used.

There are several types of suprahyoid infections; however, the author, for this discussion, has chosen the following three: pharyngomaxillary, masticator and Ludwig's.

A brief discussion for the purpose of anatomic orientation and terminology will be followed by a clinical report of cases which are characteristic of infection in each area.

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‡Original drawings by Mr. Ted Bloodhart, Los Angeles, Calif.

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PHARYNGOMAXILLARY.

When tonsillar or pharyngeal disease extends laterally through the superior constrictor muscle it enters the pharyngomaxillary fossa which was described by Mosher[1] in 1929 (see Figs. 1 and 2). A study of the diagrammatic sections

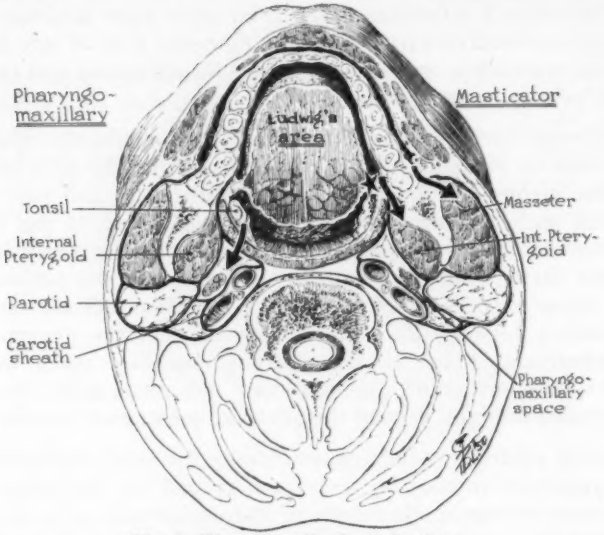

Fig. 1. Diagrammatic Cross Section.
Pharyngomaxillary area and relationships shown on left. Arrow indicates course of disease through the superior constrictor muscle.
Masticator space is shown on right with arrows indicating course of dento-alveolar disease.
Star indicates area of spontaneous drainage in masticator abscess.

reveals the anatomic basis for the signs and symptoms which may result from disease in this area. The tonsil, if present, and the lateral pharyngeal wall are pushed toward the midline. Pressure laterally against the internal pterygoid muscle results in a certain degree of trismus, while external swelling over the parotid is not infrequent. Should the process reach the inferior limit of the space, at the level of the hyoid bone, induration, swelling and tenderness below the angle of the mandible will result. Most important, however, is the intimate relationship to the contents of the carotid sheath. In addition to a periphlebitis, or perijugular abscess, two other very seri-

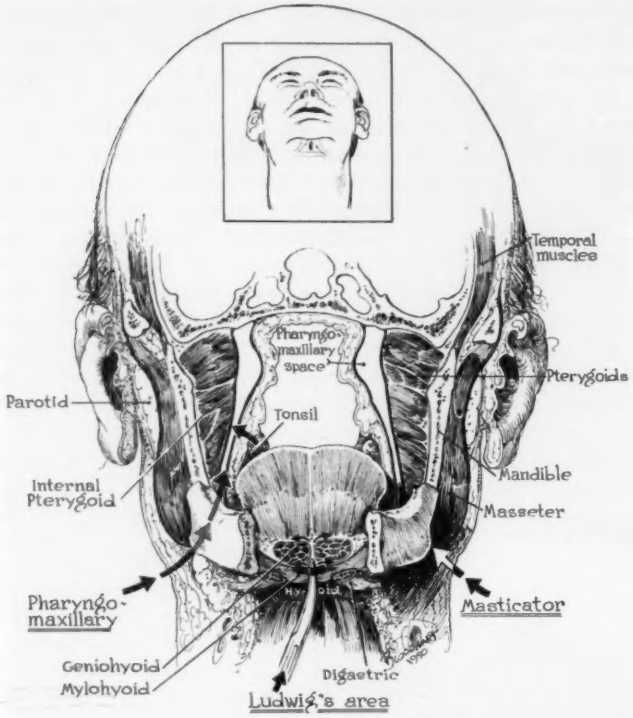

Fig. 2. Diagrammatic Frontal Section.

Pharyngomaxillary area is shown on left with arrows indicating the space to be medial to internal pterygoid muscle.

Masticator space (musculofascial sling) shown on right with muscles of mastication and ascending ramus of mandible.

Ludwig's area shown in midline with important muscles.

Insert shows midline incision for Ludwig's.

ous complications may occur. The first is an endophlebitis of the internal jugular vein and its tributaries, the second is the dreaded erosion which most commonly involves the internal carotid artery.

MASTICATOR.

Dentoalveolar and subperiosteal infections of the mandible have so frequently extended to involve the masseter and inter-

nal pterygoid muscles that this area has also become an entity. It is known as the masticator space and was described by Coller and Yglesias[2] in 1934 (see Figs. 1 and 2). A review of the diagrammatic sections reveals this space to be the immediate lateral neighbor of the pharyngomaxillary space; therefore, the signs of disease in this area closely resemble those of pharyngomaxillary involvement. On frontal section (see Fig. 2) this area appears as a musculofascial sling containing the muscles of mastication and the ascending ramus of the mandible. Posterior extension of disease from the molar teeth, most commonly the third molar, may be either along the medial or along the lateral aspect of the mandible. Immediately upon reaching the masseter or pterygoid muscles, definite trismus occurs. The predominance of internal or external swelling will depend upon whether the pathology is most marked along the lingual or along the buccal margin of the mandible. Infection in the masticator space often produces, in addition to extreme trismus, induration involving the posterior sublingual tissues and the lateral pharyngeal wall.

LUDWIG'S.

The term Ludwig's angina is so permanently implanted in our medical literature that it may appear academic to be controversial regarding the term; however, due to its varied and often promiscuous usage, it becomes imperative that one state what he considers to be Ludwig's and what anatomic area is involved. The author agrees with the very concise and definite description of Ashurst.[3] He stated, "It is an acute inflammatory process involving the cellular tissues of the floor of the mouth and the submaxillary region of one or both sides of the neck. It is the simultaneous involvement of the submaxillary and the sublingual tissues in a confluent septic cellulitis that warrants the condition being recognized as a distinct clinical entity." He further stated that it may begin in either the sublingual or the submaxillary area, but until it involves both it does not constitute Ludwig's. The term "submaxillary region" should be emphasized, for this is not primarily a

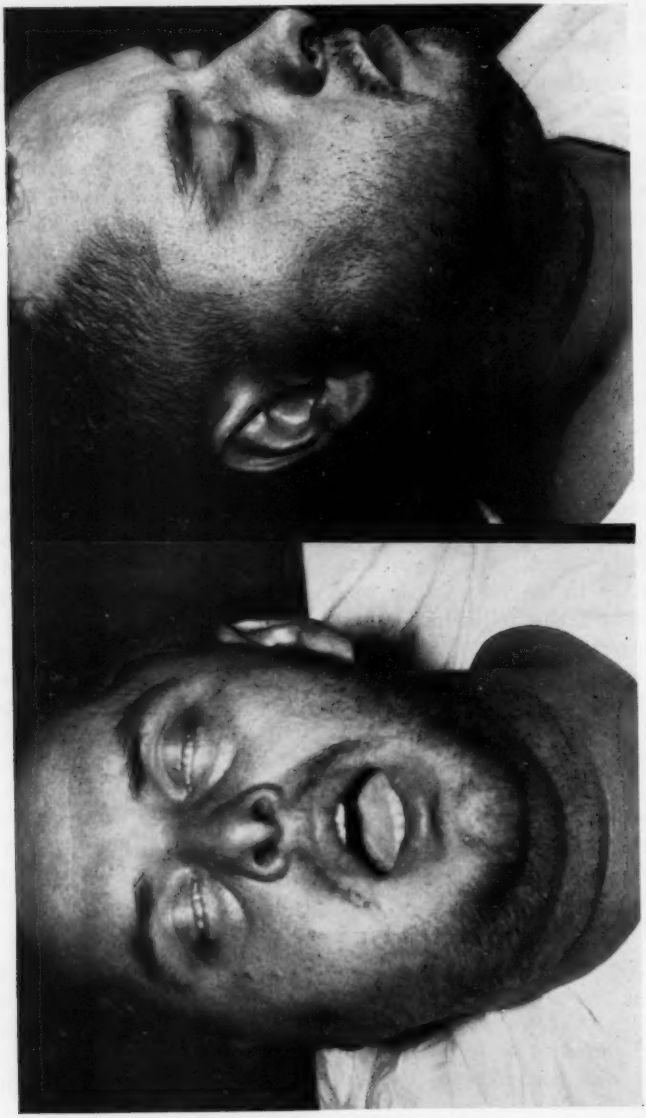

Fig. 3. Pharyngomaxillary Abscess. Upon admission to hospital.

submaxillary gland infection. Infections of this type are usually of dental origin.

The thin mylohyoid, because of its wide bony insertions, presents a formidable muscular diaphragm (see Fig. 2). Above it lie the thick geniohyoids and the muscular tissues of the tongue itself. A study of this musculofascial area with its unyielding attachments to the mandible and to the hyoid bone readily reveals how a spreading cellulitis may produce alarming symptoms due to respiratory embarrassment, tissue necrosis, sepsis and possibly to carotid sinus involvement.

PHARYNGOMAXILLARY ABSCESS.

The first case to be reported is that of a pharyngomaxillary abscess. This white male, age 35, complained of a mild soreness in the right side of the throat on Aug. 24, 1949. The following day he was symptom-free and remained so until Aug. 28. Upon this date, four days following the mild soreness, there was extreme pain in the right throat, there was rapidly increasing swelling about the parotid and the mandibular angle, and there was intense malaise. He was admitted to the hospital on this same day, where examination revealed the following important signs: temperature 105°, leucocytosis 26,000, moderate trismus, generalized swelling over the right side of the face and below the mandible (see Fig. 3) and marked medial displacement of the right tonsil and pharyngeal wall. There was no acute pharyngeal inflammation. The right hypopharyngeal wall was displaced medially to such an extent as to produce respiratory embarrassment. On Aug. 29, five days after the initial symptom, and one day following the marked symptoms, a preliminary tracheotomy was performed. With an adequate and safe airway established, pentothal anesthesia was administered for the incision and drainage.

A transverse incision was made at the level of the hyoid bone beneath the angle of the jaw. After cutting through the edematous subcutaneous tissue the cervical fascia was exposed and found to be very tense. When this fascia was incised no pus was encountered, *but* upon inserting a curved forceps

upward medial to the mandible, foul pus escaped under pressure (see Fig. 4). A Penrose drain was then inserted to the

Fig. 4. Pharyngomaxillary Abscess. Arrows indicate location of pus and course of Penrose drain.

Fig. 5. Pharyngomaxillary Abscess. Left, 24 hours postoperatively. Right, three weeks postoperatively.

depth of the wound. Intensive antibiotic therapy was continued and the patient made a rapid and uneventful recovery (see Fig. 5).

The important points to be emphasized are: pharyngeal history, pharyngeal wall displacement without acute pharyngeal inflammation, rapidly increasing and severe clinical picture, only moderate trismus, easy depression of the tongue on both sides posteriorly, no fluctuation, and the finding of pus medial to the jaw and to the internal pterygoid muscle.

Fig. 6. Masticator Space Infection. Upper, prior to external drainage. Right photo shows patient attempting to open her mouth. Lower, 48 hours following external drainage.

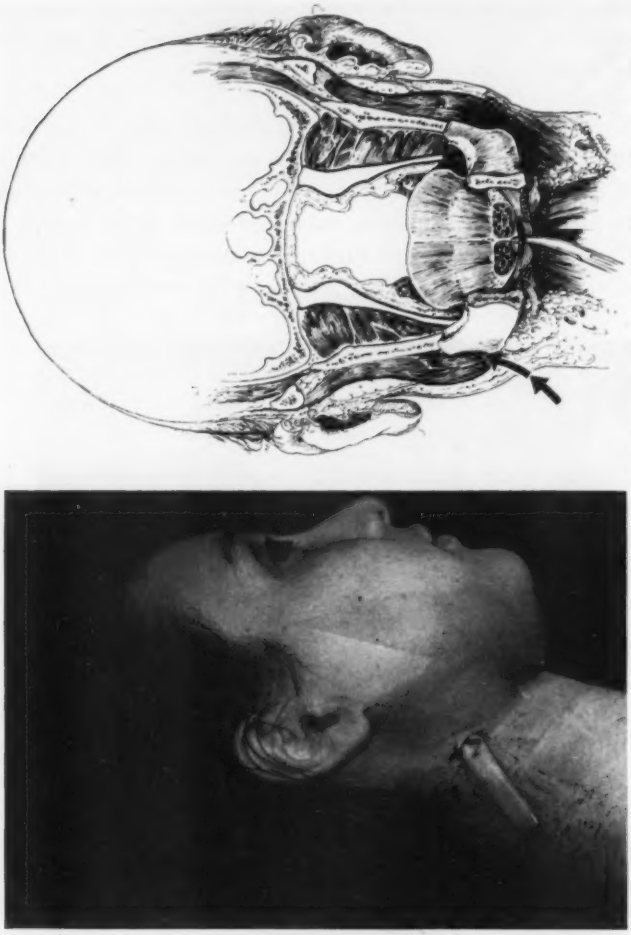

Fig. 7. Masticator Space Infection. Arrows indicate location of pus and position of Penrose drain.

MASTICATOR SPACE INFECTION.

The second patient presented disease in the masticator space.

This girl, age 20, was first seen on Jan. 29, 1949, and presented a painful swelling over the right jaw associated with trismus of moderate de-

gree, and temperature of 101.5°. Two weeks previously she had complained of a right-sided toothache which subsided following spontaneous drainage into her mouth. On Jan. 31, two days after admission, an intraoral incision was made along the mandible opposite the right lower third molar. Pus was recovered, and she was discharged on Feb. 2.

Two week later, Feb. 15, she was readmitted with marked trismus, difficult depression of the tongue on the affected side posteriorly, and tender swelling over the jaw externally (see Fig. 6). There was no fluctuation. External incision was made below the angle of the jaw and when this wound was explored *to the bone*, pus was recovered (see Fig. 7). Dental radiographs revealed an apical abscess of the right lower third molar. On Feb. 28 the offending third molar was extracted. One month following the surgical drainage the trismus and swelling had entirely subsided. It should be remembered that subsidence of trismus in these masticator cases is usually rather slow.

The important points to be emphasized are: dental history, extreme trismus, the posterior portion of the tongue on the affected side was impossible to depress, no fluctuation, the patient was not acutely ill as compared to pharyngomaxillary abscess, and the abscess was *on the bone*.

Intraoral incisions along the mandible are sometimes sufficient, but when they prove otherwise, as in this case, external drainage must be performed. In masticator abscess the pus is on the bone rather than medial to the pterygoid as in pharyngomaxillary abscess (see Fig. 2).

A large percentage of masticator space infections drain spontaneously within 10 days of the swelling, as reported by Hall and Morris.[4] When this occurs pus can be seen escaping from an area between the base of the tongue and the mandible (see Fig. 1).

LUDWIG'S.

The third patient, age 32, presented what we considered to be Ludwig's infection.

Her right lower third molar was extracted on May 16, 1949. This was followed by marked external swelling within 48 hours. A dentist performed a submental incision at this time and penicillin was administered. She entered the hospital on May 23. She was acutely ill. The temperature was 104°, and there was intense board-like swelling in the entire submaxillary region (see Fig. 8). The tongue was extremely swollen and pushed upward against the palate (see Fig. 9), and the oral floor was board-like. There was no fluctuation. There was dyspnea, dysphagia, dysphonia and drooling. The incision was immediately extended upward to the floor of the mouth. A small curved forceps was used to separate the

tissues on either side of the wound. Only a scant amount of thin secretion was recovered, although some days later there was a moderate purulent discharge. Intense antibiotic therapy was continued. The symptoms rapidly diminished, and the patient was discharged one week later (see Fig. 8).

Fig. 8. Ludwig's Infection. Upper, upon admission to hospital. Coexistence of disease in sublingual and submaxillary regions. Lower, one week later upon discharge.

The photographs (see Figs. 8 and 9) demonstrating the coexistence of disease in the sublingual and submaxillary regions appear to be typical of Ludwig's infection as we know it. No doubt there have been more fulminating cases, but the anatomy, the etiology, the signs and the treatment remain the same.

We prefer an incision similar to that described by Dr. August Beck[5] in his complete work on Deep Neck Infections.

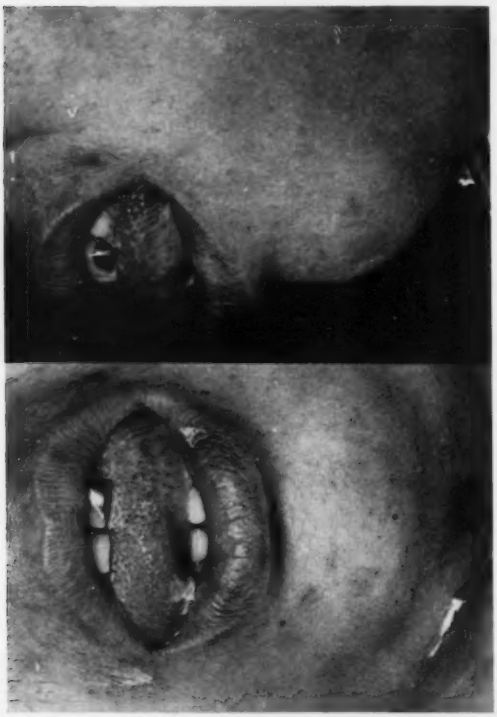

Fig. 9. Ludwig's Infection. Tongue fills the mouth.

A midline incision is made between the mandible and the hyoid bone. It is then carried upward between the digastrics and through the mylohyoid. The incision is extended upward between the geniohyoids with separation of the inflammatory tissues on either side of the midline with curved forceps for the purpose of decompression and drainage (see Fig. 10). This procedure has been effective in a large number of cases with prompt relief of the respiratory and other symptoms. There is no objection to performing a preliminary tracheotomy, after which a general anesthetic may be employed. Early recognition of Ludwig's is imperative, for there are many who feel that early decompression is essential as a preventive of tissue necrosis.

The cases reported demonstrate three important points which may be applied to all deep infections of the neck:

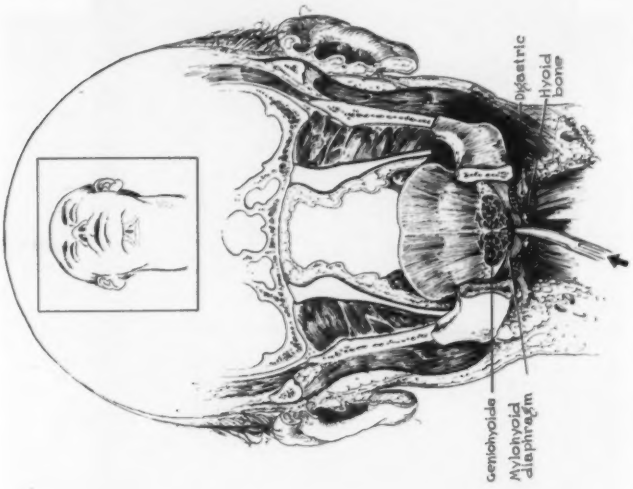

Fig. 10. Ludwig's Infection. Arrows indicate course of forceps in entering and decompressing the cellulitic tissues. Insert shows midline incision.

1. Antibiotic and chemotherapy should be used from the onset, but these agents are no substitute for drainage. Should serious symptoms supervene, surgical drainage or decompression must be performed.

2. Fluctuation is important when present, but should never be awaited.

3. An adequate airway is essential. If a safe airway is present, or can be established, a general anesthetic may be administered.

I am quite aware that there are all stages, degrees and variations of the types of infections reported above. Whether incipient cases are recognized as such is of little importance as long as the patients recover.

When the anatomy of the pharyngomaxillary, masticator and Ludwig's areas is applied to the signs, symptoms and treatment of the cases reported, it appears that each is characteristic of infection in its respective area.

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THE MANAGEMENT OF NASOPHARYNGEAL FIBROMAS.*

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In previous reports on nasopharyngeal fibromas the successive changes made in the methods of treating these tumors and the greater safety and effectiveness of these measures have been discussed. One of these communications presented the experience of our colleagues, and one of us (Figi) in dealing with 63 cases of this type encountered at the Mayo Clinic from Jan. 1, 1910, through Dec. 31, 1939. The outstanding observation noted in this study was that in 73 per cent of the cases the tumors completely disappeared before the patients attained the age of 20 years, which is approximately the time when fibromas of this type may be expected to show initial evidence of spontaneous regression. From Jan. 1, 1940, through Dec. 31, 1949, 51 additional cases of nasopharyngeal fibromas came under our observation, and these form the basis for the present study; however, the remarks and conclusions which follow reflect our overall experience with the total of 114 cases.

Nasopharyngeal fibromas, also commonly known as "juvenile basal fibromas of the nasopharynx," "fibroid tumors of the base of the skull," "fibroangiomas of the nasopharynx" and "juvenile nasopharyngeal fibromas," are uncommon in spite of this rather large group of cases. Shaheen,[1] in 1930, reported 58 cases under the title of "nasopharyngeal fibromas"; however, in view of the extremes in age of some of the patients in his series, which ranged from two to 70 years, it

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seems obvious that a number of these lesions were not of this nature. Recently Härkönen and Malmio[2] reported 31 such cases from Stockholm, and Martin, Ehrlich and Abels[3] presented a study of 29 cases seen on their service at the Memorial Hospital in New York. With these exceptions, practically every series of fibromas of the nasopharynx recorded in the literature has comprised fewer than 10 cases; accordingly, the development of present-day therapy has necessarily come from the accumulated experience of different observers.

The onset of these fibromas is definitely limited to the period of adolescence, and this is one of their most striking features. Fibrous tumors presenting somewhat the same appearance, but which usually do not behave like true juvenile nasopharyngeal fibromas, are occasionally seen in the nasopharynx, nasal fossae and accessory sinuses of patients who have not yet reached the age of puberty, and also in young adults, and in persons of advanced years. Confusion has resulted in a number of instances, for such lesions have at times been reported in the literature as juvenile basal fibromas. Since they are quite innocuous in comparison with the highly vascular, locally invasive lesions under consideration, it is essential that they be differentiated.

When the patient who has a juvenile nasopharyngeal fibroma attains sexual maturity, or roughly at about the age of 20 to 25 years, the tumor generally becomes less active than before and shows a varying degree of natural regression; however, as stated in a previous communication, we have never observed the complete spontaneous disappearance of these fibromas that is so often referred to in the literature. At the same time, it should be noted that because of the locally destructive character of such tumors, and their proximity to a number of vital structures, we have not felt justified in marking time and idly observing them to see whether complete recession would occur; moreover, most of our patients who had nasopharyngeal fibromas have lived at considerable distances from the clinic, and their finances have been too limited to permit them to return for observation more fre-

quently than the situation demanded. Rarely these growths do persist and continue to progress after the patient reaches sexual maturity. Included in the present series is a man who came under our care at the age of 28 years with an extensive basal fibroma of the nasopharynx and right nasal fossa that had first produced symptoms at the age of 23 years and which had recurred following repeated previous removal.

The predominance of these tumors in males and their association with adolescence have naturally given rise to hypotheses concerning a probably etiologic relationship with a disturbance of sexual development. Actually, however, little more is definitely known about their cause than that of neoplasms of other types.

Juvenile basal fibromas generally grow slowly through the period of sexual development, although there may be intervals of markedly accelerated activity due to an acute inflammatory process, trauma, or for no obvious reason. While these neoplasms are perfectly benign histologically, and never metastasize, they may be highly destructive locally and often produce severe debility. Because of their proneness to cause pressure absorption of bone and to send out processes which invade and enlarge normal ostia and bony fissures, any one or all of the nasal accessory sinuses, the retromaxillary fossa, the orbit, and even the cranial cavity may be invaded.

As has been repeatedly emphasized by writers on this subject, the tumors under consideration occur predominantly in males; in fact, the present group of 51 patients consists entirely of males, and Martin[3] and his colleagues, from their study of 29 cases, voiced the conviction that the neoplasm never occurs in females; however, the personal series of 63 cases reported 10 years ago included five females, and a recent critical review of these convinces us that they definitely belong to this category. Other authors also have noted the infrequent occurrence of these tumors in females. Thomson and Negus[4] stated that they are rare in girls, and one of the illustrations in their textbook is a photograph of a young Negress with a tumor of this type protruding from the nostril and

producing the characteristic "frog-face" deformity often associated with advanced neoplastic involvement of the nasal fossae.

Among the 51 patients reported here, the age of onset, with one exception, ranged from nine to 17 years, averaging 13.6 years; the age at the time of examination ranged from 10 to 20 years, averaging 15.1 years; and the age at which the tumor was arrested ranged from 10 to 21 years, averaging 16.9 years (see Table 1). In the one exception referred to

TABLE 1.
NASOPHARYNGEAL FIBROMAS. AGE OF PATIENTS.*

Age	Range, Years	Average Years
At time of onset	9-17	13.6
When first seen at clinic	10-20	15.1
When tumor arrested	10-21	16.9

*Exception: One patient not included, whose symptoms began at age 23 years.

previously, symptoms had first been noted about the age of 23 years, and the tumor was continuing to progress actively at the time of our examination at the age of 28 years. There was not the slightest doubt in our minds concerning this tumor's being a juvenile basal fibroma, and the experienced laryngologist who had treated the patient previously was of the same opinion. Depending upon the site of origin and the rate of growth, it seems likely that the lesion may remain symptomless and entirely silent for a period ranging from a few months to several years. In the present series the patients had complained of symptoms, or signs had been observed by others, for from one month to several years, averaging 18.2 months prior to our examination. An interesting observation in this connection is that reported by Martin and his associates of an asymptomatic nasopharyngeal fibroma discovered in a boy aged 15 years during routine physical examination. The tumor was observed by them for two and one-half years, and they noted no appreciable change in its size. Of interest here also is a symptomless lymphosarcoma of the nasopharynx

seen at the Mayo Clinic some years ago in a woman 61 years of age.

The clinical findings in cases of nasopharyngeal fibroma are quite uniform. Examination with a nasopharyngeal mirror shows a hard, rounded or slightly nodular, deep red, purplish or grayish tumor filling a varying portion of the nasopharynx and often extending into one or both nasal fossae as well. A striking feature is the marked vascularity of the overlying mucous membrane, numerous prominent tortuous vessels generally being evident. Ulceration of the mucous membrane is unusual unless there has been recent trauma. The attachment is usually broad, sessile and firm, and the mass generally is quite immobile. Rarely the tumor has a definite pedicle, and its lower portion infrequently is visible below the palatal border. Secondary attachments may be present if previous attempts at removal have been carried out. Anterior rhinoscopy usually shows a varying portion of one nasal fossa filled with neoplastic tissue, which at times crowds the septum well over. The mass may be present in the nasal vestibule or even protrude from the nostril (see Fig. 1). There may be broad-

Fig. 1. Recurring juvenile fibroma of left nasal fossa and nasopharynx, presenting in the left nostril.

ening of the nasal bridge and root, bulging of the cheek, or elevation and proptosis of the eye, due respectively to involvement of the upper part of the nasal fossa, the ethmoid labyrinth, the antrum, the retromaxillary fossa or the orbit.

In a previous report on this subject, it was stated that fibromas of the nasopharynx provide one of the few exceptions to the generalization that biopsy should be made in all cases of neoplasm occurring in the upper part of the respiratory tract. Our feeling concerning this is unchanged. An experienced laryngologist who has seen a few nasopharyngeal fibromas can, as a rule, recognize them quite definitely on clinical examination. The gross appearance of the tumor as seen in the nasopharyngeal mirror, or on direct inspection of the nasal fossa, is very characteristic; however, if the lesion is growing actively, is recurring following recent removal, or is acutely inflamed, the clinical picture may be highly suggestive of a malignant neoplasm, and biopsy becomes essential. When any question has existed in this regard, we have routinely performed biopsy in these cases; otherwise it has been avoided. In the present series biopsy was performed in a little more than one-half of the cases. If the diagnosis is obvious from gross inspection, it is advantageous to refrain from removal of tissue for microscopic study, as this procedure often gives rise to profuse bleeding which is difficult to control, either by firm packing or by electrocoagulation. At times performance of a biopsy has resulted in such serious loss of blood that ligation of the external carotid artery and transfusion have been required, and removal of the tumor has been rendered much more difficult. In one of the patients in this series electrocoagulation was rendered impossible by the profuse bleeding induced by biopsy, and ligation of the external carotid artery and transfusion were necessary.

Malignant change in nasopharyngeal fibroma has been reported by some observers, and Thomson and Negus[4] considered this a possibility. Until recently we have been skeptical concerning the likelihood of this occurrence; however, we have recently had under observation a man, 20 years of age, from

whose nasopharynx, nasal fossae and sphenoid sinus we removed a recurring vascular fibroma, 4 cm. in diameter, three and one-half years ago. This patient has at no time presented obvious recurrence of his tumor, but for the past two and one-half years has at intervals experienced moderately severe right frontal and parietal headaches. During recent months these had become increasingly severe and right external rectus palsy and dimness of vision developed, which progressed to total blindness of both eyes. Roentgenograms showed what appeared to be malignant destruction anterior to the sella. The neurologic consultants on our staff were confident that the condition was malignant, and considered it inoperable; however, the possibility of an inflammatory process could not be definitely excluded.

The treatment of nasopharyngeal fibromas has undergone considerable change during the past few decades. Primarily therapy was entirely surgical, but experience generally with such measures was a high mortality rate and frequent recurrence necessitating repeated operations. Consequently, observation of the effectiveness of radiation in dealing with these lesions resulted in its almost complete replacement of surgical procedures for a period. More recently the combination of surgical diathermy and radiation, the latter preferably used interstitially, has been found to produce the most satisfactory results. Surgical exposure of the tumor frequently is necessary in order to permit applying these measures with greatest effectiveness.

In all but nine of the 51 patients in this series, the fibromas had either been removed surgically, or treated with radiation, or some surgical diagnostic procedure had been carried out prior to our examination (see Table 2). Removal surgically or with electrocoagulation or both had been carried out in 28 patients on from one to six occasions. Nine had received interstitial radiation from one to four times; 15 had been given Roentgen therapy, either alone or combined with surgical measures or interstitial radiation. One patient had received five courses of Roentgen treatment, and another had been given 80 applications of Roentgen radiation. What was

TABLE 2.
NASOPHARYNGEAL FIBROMAS. PREVIOUS TREATMENT.

Cases in this series	51
Cases without previous treatment	9
Cases with previous treatment*	42
Surgical removal or electrocoagulation or both	28
Interstitial radiation	9
Roentgen therapy	15
Biopsy only	3
Sclerosing injection	1

*Several patients had been treated by surgical removal and electrocoagulation, as well as by interstitial radiation.

assumed to be some type of caustic or sclerosing solution had recently been injected directly into the tumor in one case. Another patient who came with a huge mass obstructing both nasal fossae complained of residual diplopia dating from an attack of meningoencephalitis that had followed the last surgical removal of his tumor some months previously. In three cases biopsy only had been carried out. In one of these, in which the procedure had been done a week previously, the patient came with firm gauze packs in the nose and nasopharynx. He had bled so severely that he had severe secondary anemia, and it was necessary to admit him directly to the hospital for a transfusion.

Recent surgical procedures generally increase the difficulty of subsequent operative measures and render them less effective. This is due to the fact that they tend to obscure the limits of the residual tumor, decrease its mobility, and often increase its vascularity by producing multiple secondary attachments. On the other hand, intensive Roentgen therapy at times appears to render subsequent treatment, especially with interstitial radiation, more effective. It has seemed in some instances to facilitate electrocoagulation, probably by reducing the blood supply. At least in a number of cases in this group in which the patients had previously received heavy Roentgen treatment, fewer treatments were required to eradicate the fibroma than were necessary in some of the others.

The surgical approach to nasopharyngeal fibromas has a direct bearing on the possibility of complete removal, and

accordingly on the prognosis. It necessarily varies with the extent, activity, vascularity and situation of the growth, the nature, site, and area of its attachment, and the previous treatment. Adequate exposure is essential, but often it is difficult to obtain without mutilation, which rarely is justifiable. Such procedures as splitting the soft palate and temporary resection of the maxillae in order to gain access to the tumor have gone into the discard. The approach may make the

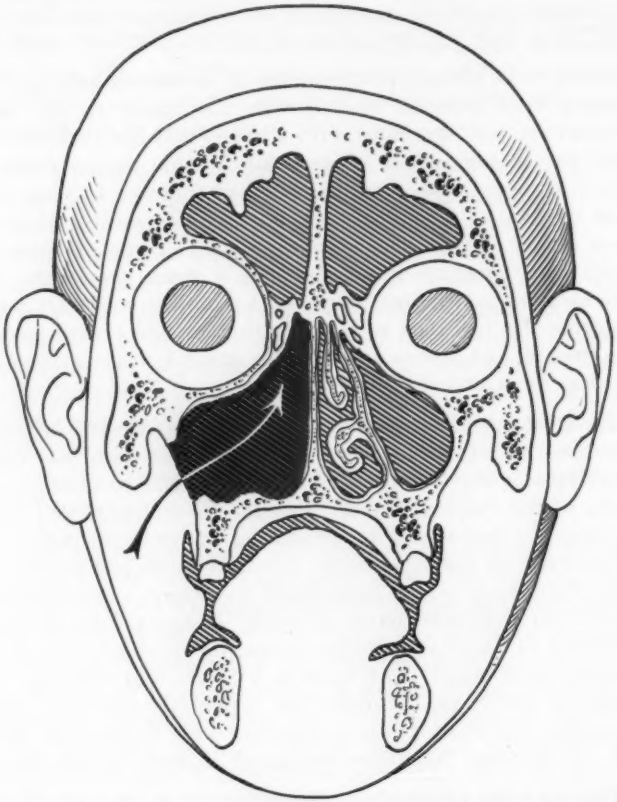

Fig. 2. Schematic drawing showing complete removal of the nasoantral wall during course of transantral approach to tumor of the nasal fossa or nasopharynx.

difference between an extremely hazardous and a reasonably safe procedure. It may permit eradication of the growth in a single operation with a brief convalescence, or necessitate repeated surgical measures or applications of radiation during the course of a number of months or even several years with a much less satisfactory ultimate result.

In the group of nasopharyngeal fibromas reported in 1941, treatment directly to the tumors was carried out entirely through the nostrils and transorally through the nasopharynx. In the present series also a majority of the tumors were dealt with in the same manner; however, during the past three and one-half years a transantral approach has been used to secure access to the neoplasm in a number of these cases, and the procedure has proved highly effective. It entails performing a Caldwell-Luc operation, most of the anterior wall of the antrum being removed, and then taking away a varying portion of the lateral wall of the nose, depending upon the situation of the growth (see Fig. 2). The approach through the nares and nasopharynx usually affords quite satisfactory access when the fibroma is situated in the lower part of the nasal fossa and in the nasopharynx. Better exposure of these regions can be secured transantrally, and this is true also when the tumor is in the superior portion of the nasal fossa, the ethmoid labyrinth, antrum, sphenoid sinus, orbit or retromaxillary fossa; in fact, involvement in many of these situations is inaccessible through the nostrils and nasopharynx and removal by these routes is much less certain and bears infinitely greater hazard than by the transantral approach (see Fig. 3). Many nasopharyngeal fibromas have a broad sessile attachment high on the posterior wall or over the vault of the nasopharynx, and a surprising number are attached on the posterior portion of the lateral wall of the nose, especially high up. All of these regions are readily accessible through a transantral approach, as is also the involvement commonly extending from these regions into the regions mentioned previously; that is, the sphenoid sinus, orbit, and retromaxillary fossa. At times the maxillary sinus is not actually invaded by the tumor, yet the antral cavity is encroached upon and

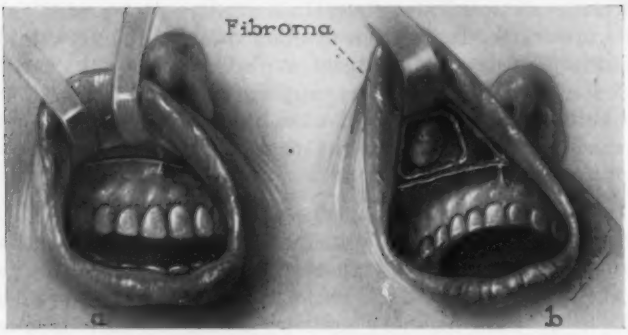

Fig. 3. Transantral approach for removal of juvenile basal fibroma of the nasopharynx and right nasal fossa. The anterior tip of the tumor is presenting in the midportion of the nasal fossa.

reduced in size as much as 75 per cent or more, owing to the mass in the nose or in the pterygomaxillary fossa crowding upon its mesial or posterior walls. Even when this is true, excellent exposure usually can be secured through the antrum.

A vulcanite nasal speculum is used when the diathermy is applied through the nostrils in order to avoid injury to these structures. For coagulation of that portion of the tumor which is situated in the nasopharynx the soft palate is retracted by means of a rubber catheter introduced through either nostril and brought out through the mouth, as described in a previous communication (see Fig. 4). Intravenous pentothal sodium anesthesia is employed in removal of these tumors, and an intratracheal tube is inserted through the mouth to maintain the airway, and for the administration of oxygen. A moist gauze pack placed firmly about this tube holds it to one side out of the line of vision and prevents gravitation of blood into the lower part of the respiratory tract. In some cases, because of the activity, extreme vascularity and extent of the tumor insertion of radium alone through the nostrils and nasopharynx has been carried out, rather than an attempt made at surgical removal. With extension of the growth well laterally in the retromaxillary fossa and bulging of the cheek and soft palate, implantation of radon seeds is at times made

directly through the posterior portion of the buccal fold and through the soft palate.

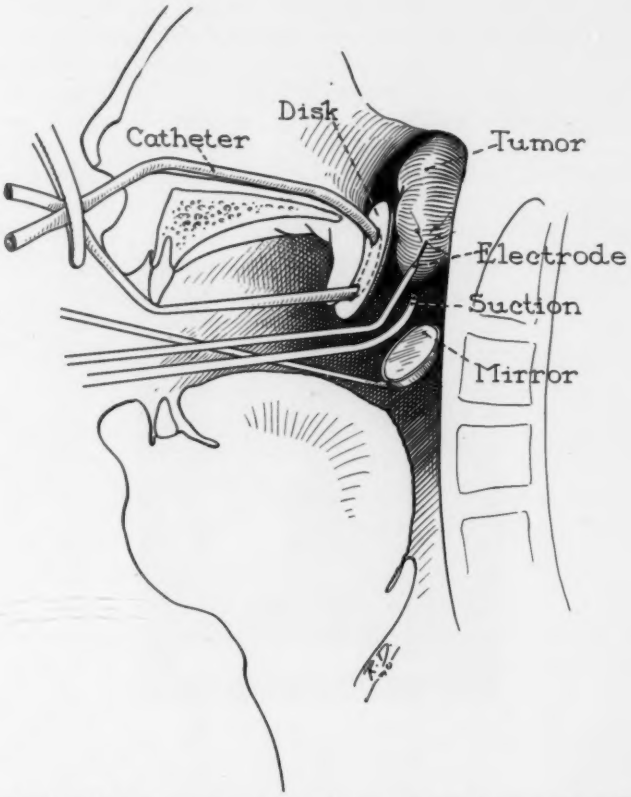

Fig. 4. Method of exposing portion of tumor in nasopharynx. (From Figi, F. A.: Fibromas of the Nasopharynx. Jour. A. M. A., 115:665-672, Aug. 31, 1940.)

Much the same procedure is followed when the tumor is approached transantrally except that the better exposure usually obtained permits more accurate determination of the limits and relationships of the tumor and facilitates its removal. Although many of these tumors are quite firm and

present as a discrete, rounded, nodular mass, some of them are quite soft and friable, and the capsule is thin and delicate and tears readily on slight traction. Others present accessory lobules or processes of varying size which become detached easily, rendering clean removal difficult, and often giving rise to recurrence. Providing hemostasis is reasonably satisfactory, as the electrocoagulation progresses under direct transantral exposure, gentle traction on the diathermy electrode, which tends to adhere to the tumor, frequently will bring the encapsulated surface of the mass into view, and thereby assist greatly in its clean removal. At times also blunt dissection is helpful in this connection (see Fig. 5). These measures are

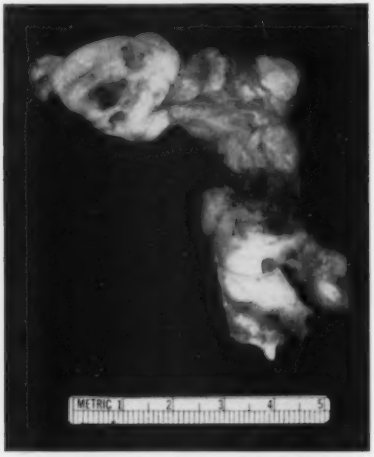

Fig. 5. Juvenile basal fibroma of the right nasal fossa, nasopharynx, sphenoid sinus and retromaxillary fossa, which was removed through a transantral approach. The tumor had a broad sessile attachment on the posterior portion of the lateral wall of the nasal fossa.

followed by packing radium tubes or needles into the wound, even though removal appears to be complete, or by implantation of radium needles or radon seeds into any recognizable remnant of fibroma when complete surgical removal cannot be accomplished. In some of the cases taken care of in this manner it is possible to eradicate the tumor in a single operation, which rarely is feasible when an extensive active fibroma is approached through the nose and nasopharynx; however,

even with adequate exposure complete removal in one operation may not be possible, and residual tumor or further growth of a remnant of it must be taken care of later.

While active bleeding commonly occurs from fibromas of the nose and nasopharynx, severe secondary hemorrhage from the operative wound does not take place as often following removal by diathermy as one might well expect. Bleeding sufficiently severe to necessitate ligation of the external carotid artery was encountered in only two cases in this series. In view of this, we do not deem it advisable to perform ligation of the external carotid artery routinely as a preliminary measure. Ligation of the external carotid artery on the side of the principal involvement was carried out immediately preceding transantral exposure of the tumor in five cases in this series. It was our impression that this was of material assistance in controlling the hemorrhage, although bleeding was profuse in some cases, in spite of this procedure. Bilateral ligation of the external carotid arteries probably would have helped considerably more, and one need have no hesitancy in carrying this out when it is indicated. When ligation is performed, it should be at the same time as the actual removal of the growth in order to be most effective. Collateral circulation often is established so promptly that the lapse of a few days is likely to lessen its effectiveness greatly. It probably would be of no permanent benefit in preventing the severe bleeding at times associated with the crusting resulting from treatment.

Secondary operations usually are best carried out after an interval of six weeks to two months, but in a few instances we have performed the second stage of an operation after approximately two weeks. This was required in two of the cases in this group. One of these was rendered necessary by the fact that the patient reacted badly during the initial operation, the pulse becoming very rapid and barely palpable, and there being no response to a transfusion given on the table. Another patient requiring a two-stage removal presented an extensive, partially necrotic tumor filling the right nasal fossa, antrum, ethmoid labyrinth and sphenoid sinus,

and extending into the retromaxillary fossa and nasopharynx. With the exception of its retromaxillary portion, this rapidly growing fibroma appeared to have been completely removed at the first operation (see Figs. 6A and 6B). Since there had

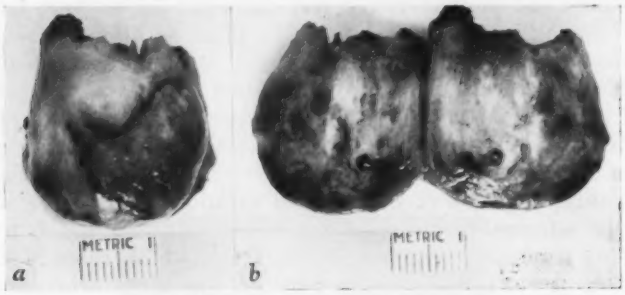

Fig. 6A. Encapsulated fibroma of the nasopharynx. This portion of the tumor was situated in the nasopharynx and dropped down into the hypopharynx upon transantral electrocoagulation of its pedicle. It comprised only a small part of the entire fibroma, which filled the right nasal fossa, antrum, ethmoid labyrinth, sphenoid sinus and nasopharynx of a lad 15 years of age. (B) Tumor opened up to show its homogeneous character.

been a good deal of bleeding during the procedure, and the operation had been rather protracted, it seemed inadvisable to prolong it further. It was terminated, and radium points were inserted into this remnant; however, in spite of the interstitial radiation, about 10 days later what looked like actively recurring fibroma appeared in the lower posterior part of the maxillary sinus. This was explored through the antrum on the twelfth postoperative day, and an encapsulated lobulated tumor measuring 7 by 4.5 by 3 cm., which microscopically proved to be a vascular fibroma, was removed by blunt dissection, and the wound was lightly electrocoagulated (see Fig. 7). Nineteen days later there was severe bleeding from the wound, probably due to the giving way of the internal maxillary artery, and ligation of the external carotid artery became necessary. This patient, 15 years of age, is now in robust health and free from tumor after 20 months.

The effectiveness of therapy for nasopharyngeal fibromas often is difficult to evaluate because so many factors must be taken into consideration. Important among these are the age

Fig. 7. Residual encapsulated lobulated fibroma removed from the posterior part of the right antrum and retromaxillary fossa 16 days after the main portion of the growth, including the mass shown in Figs. 6A and 6B was taken out. This portion of the tumor measured 7 by 4.5 by 3 cm.

of the patient, the accessibility of the tumor, its activity, size, vascularity, site of attachment, previous treatment, and its conformation; that is, whether it consists of a compact mass or of multiple lobules. Allowing for the variation in size and activity of the tumors, the length of time that therapy had to be continued and the number of treatments required in each case probably are the most accurate criteria of the potency of treatment.

Of the 51 patients in this series, one did not stay for treatment. Forty-two received electrocoagulation and interstitial radiation (see Table 3). This was applied through the nostrils and nasopharynx in 33 cases, through the antrum in nine, and through a nasofrontal approach in two. Several of these 42 patients were treated through more than one approach at different times. One patient was treated by electrocoagulation only. Seven patients received interstitial radiation only, radium needles or radon seeds being implanted through the nostrils and nasopharynx. In two of these seven

TABLE 3.
NASOPHARYNGEAL FIBROMAS. METHODS OF TREATMENT.

		Number Treated
Electrocoagulation and interstitial radiation	*	42
Nasal and nasopharyngeal approach	33	
Transantral approach	9	
Nasofrontal approach	2	
Electrocoagulation only		1
Interstitial radiation only		7
Total		50
Supplemental procedures		
Radium packs		3
Primary external carotid ligation		5
Secondary external carotid ligation		2
Total		10

*Several patients were treated through more than one approach at different times.

patients implantation of radon seeds was also made through the soft palate, and in one through the cheek, the lateral wall of the nose and directly through a bony defect in the hard palate into the mass that was perforating at this point.

In 10 cases supplemental procedures were used. In five cases primary, and in two cases secondary, ligation of the external carotid artery was performed. Three patients with extensive diffuse recurring fibromas received external radiation with radium packs in addition to the therapy mentioned previously; however, it is our feeling that radiation either with Roentgen therapy or with radium packs is much less effective in controlling these fibromas than is radium applied to the surface of the growth or implanted into it. It also bears the possible danger of inducing secondary radiation changes in the skin, and since most of the patients with nasopharyngeal fibromas are in the stage of active development, the possibility of interference with normal osseous growth is ever present. In view of the foregoing considerations, the wisdom of using intensive Roentgen therapy in these cases, as is commonly practiced, may well be questioned.

Four patients in the series received treatment less than six months prior to the time of writing of this paper, and three of these have not been observed since they were taken care of. The one patient in this group of four who has returned for observation shows no evidence of tumor, but the interval has been too brief for this to be significant. No follow-up has been obtainable for seven patients treated from four to eight years ago. The prognosis appeared very favorable for some of these, and it seems likely that at least several of them are free from tumor. One patient first treated at the age of 17 years, and who is now 20 years of age and a member of the United States Air Force, presented Roentgenographic evidence of recurrence along the wing and body of the left sphenoid bone when last observed a little more than a year ago. Treatment with radium packs only was given at that time, as no clinical evidence of tumor could be made out, and it was feared more active therapy might result in injury to the eye. The patient still has occasional attacks of epistaxis, which may be due to crusting.

Thirty-six of the 39 patients for whom a follow-up was obtainable present no evidence of active fibroma (see Table 4).

TABLE 4.
NASOPHARYNGEAL FIBROMAS. RESULTS OF TREATMENT.

Cases with treatment	50
Less than six-month follow-up	4
Lost to follow-up	7
Cases with follow-up	39*
Arrested cases	36
No evidence of tumor	29
Inactive tumor present	7
Deaths	2

*One case uncontrolled when last seen.

Twenty-nine of these are entirely free from evidence of tumor. The remaining seven show residual thickening or a small nubbin which has remained inactive during periods of observation ranging from six months to five years. The tumors are under control in four (57 per cent) of the seven patients

treated entirely with radiation and in the one who underwent electrocoagulation only. The same is true also in 24 (73 per cent) of the 33 treated through the nostrils and nasopharynx, and in seven (78 per cent) of the nine treated through a transantral approach (see Table 5).

TABLE 5.
NASOPHARYNGEAL FIBROMAS. METHODS OF TREATMENT. COMPARISON OF RESULTS.

	Number Treated	Number Controlled
Electrocoagulation and interstitial radiation	42	31 (73.8%)
Nasal and nasopharyngeal approach	33	(24) (72.7%)
Transantral approach	9	(7) (77.7%)
Electrocoagulation only	1	1 (100%)
Interstitial radiation only	7	4 (57.1%)
Totals	50	36 (72%)

Among the patients who are now free from evidence of fibroma, the number of therapeutic measures necessary in each ranged from one to 11, the average being 2.7 (see Table 6). For the cases in which interstitial radiation only was

TABLE 6.
NASOPHARYNGEAL FIBROMAS. COMPARISON OF PROCEDURES USED IN ARRESTED CASES.

	Number of Procedures, Average	Length of Treatment, Years, Average
Entire series	2.7	1.1
Interstitial radiation only (4 cases)	3	1.4
Nasal and nasopharyngeal approach (24 cases)	2.9	1.6
Transantral approach (7 cases)	1.8	0.5

used, the number of treatments averaged three. For the cases in which the major portion of the therapy was carried out through the nostrils and nasopharynx, the number of treatments averaged 2.9. Among those in which it was applied practically entirely transantrally, the number averaged 1.8.

The length of time that treatment was continued prior to complete disappearance of the tumor, or until there persisted only a small, inactive remnant, ranged from six months to five years, and averaged 1.1 years. In the cases in which radiation alone was used treatment lasted for an average of 1.4 years, and the patients averaged 17.1 years of age at its conclusion. In those in which diathermy and radium were applied through the nares and nasopharynx treatment lasted for an average of 1.6 years, and the patients also averaged 17.1 years of age at its termination. In those in which surgical measures were carried out through an antral approach treatment lasted an average of six months, and the average age of the patients was 15.5 years at the time the fibroma disappeared. Contrary to the opinion of Martin and his associates,[3] expressed in their recent communication on this subject, that aggressive measures, either by irradiation or by surgery, will never completely and permanently eradicate these neoplasms, it is our firm conviction that active treatment will cure a high percentage of them. The foregoing figures strongly bear this out.

Considering the situation of these tumors and the extent of many of them, the low mortality rate associated with their removal is gratifying. There were no immediate operative deaths in the group; however, two of the patients died a few months following operation. One of them, a boy 11 years of age, in whom a very extensive recurring tumor was removed with electrocoagulation and interstitial radiation through a nasofrontal approach, returned to the clinic three months later because of severe headaches of a week's duration, and presented evidence of meningitis, to which he succumbed 12 days later. The other, a boy 17 years of age, with a recurring fibroma filling one nasal fossa, on whom we operated three times, and to whom interstitial radiation was given on two additional occasions, had a fatal hemorrhage at home four months after his tumor was last treated with electrocoagulation and interstitial radiation. This was the only other patient in the series in whom removal of the tumor was carried out through a nasofrontal approach. This is mentioned, not be-

cause the procedure had direct bearing on the fatal outcome, but because it is indicative of the high situation of the lesion.

CONCLUSIONS.

1. The most satisfactory treatment for nasopharyngeal fibromas is a combination of electrocoagulation and interstitial radiation.

2. These measures may be used through the nares and transorally through the nasopharynx, but they are more safely and effectively applied through a transantral approach with or without preliminary ligation of the external carotid artery.

3. With proper treatment a high percentage of nasopharyngeal fibromas can be completely eradicated or reduced to a small inactive remnant; in fact, by means of electrocoagulation and interstitial radiation applied transorally complete removal often can be accomplished in a single operation.

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CONGENITAL CHOANAL ATRESIA.*

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Judging from the increasing number of cases reported in the recent literature, congenital choanal atresia should be considered an unusual rather than a rare developmental anomaly. Current interest has been chiefly centered on the surgical methods of correction of the atresia, and little attention has been paid to several important features in the clinical picture. This report of additional cases of choanal atresia is made to review the effect of unilateral and bilateral total choanal occlusion on the physiology and development of the nose, sinuses and ears and to comment upon the hereditary aspects of this condition.

My experience comprises five cases of total unilateral bony occlusion of the posterior nares and one case of bilateral choanal atresia in an adult. A recent case was the infant daughter of a woman whose occlusion I had corrected five years previously. This was an unusual finding of unilateral congenital choanal atresia in two generations.

Little is noted in the literature regarding the hereditary nature of congenital choanal atresia. It is usually stated that the condition is rare, has little predisposition for associated congenital defects and has a low hereditary incidence. A survey of the available references has shown that Stewart[1] reported bilateral atresias in sisters and a history of an infant brother who died of asphyxia in infancy, probably due to the same cause. Wilkinson[2] cited Lang reporting a boy, his sister and mother, and possibly a brother and a maternal aunt of the

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mother with unilateral atresia. Phelps[3] states that there is on record a family in which the mother, her two sisters, her two daughters and a son were affected. Wilkinson[2] reported complete bilateral bony atresia in three sisters. In discussing this paper, Shea stated that Guyton had two cases, cousins, under his care. Evans[4] reported a case of bilateral bony occlusion of the choanae in a three weeks old infant. Four other children in this family died in early infancy. The history strongly suggests the presence of the same deformity. In a recent communication, Dr. J. L. Cabaniss, of Roanoke, Va., reports an interesting family under his care, consisting of a mother and twins. The mother has complete choanal atresia on the left side; one of the twins has a complete atresia on the left side and partial on the right side, and the other twin had bilateral choanal atresia operated upon by Dr. Fletcher Woodward at the University of Virginia.

Macklin,[5] after reviewing this data, states that the finding of a rare developmental condition in several members of the same family bespeaks a genetic origin; however, she points out, "the mode of transmission is not easily answered. The few families mentioned in the literature would indicate that it is a dominant factor in most instances."

The most interesting, but infrequently discussed, features of congenital choanal atresia are its relationship to the development of the sinuses, and to the physiology of the nose and ears. To illustrate how little the development and function of the nose and sinuses are affected, the following cases, one unilateral and one bilateral, are reported as characteristic of choanal atresia in an adult.

The patient, a woman age 52, stated that she had never been able to breathe or smell through the left side of her nose. It had caused her little inconvenience; occasionally a mass of mucus would drain from the left nostril, especially on flexion of her head. Her ears have never given her any trouble.

Examination showed a complete bony atresia of the left posterior nares. The mucous membrane was pale and boggy and

responded fairly promptly to the application of vasoconstrictors. The left nasal fossa was narrowed by a deviation of the septum to the left. A moderate amount of glairy, tenacious mucus was removed from the left fossa by suction. Most of the mucus was collected in a depressed recess formed in the anterior portion of the floor of the nose. Anosmia was present on the occluded side; the olfactory sense appeared slightly impaired on the opposite side. The skin of the upper lip on the left side showed areas of excoriation. The right nasal fossa appeared normal. No other abnormalities or asymmetries were noted.

Fig. 1. Unilateral choanal atresia. X-ray of sinuses demonstrating full development of maxillary and frontal sinuses.

Fig. 2. Case 1. Unilateral choanal atresia. Lipiodol in left nasal fossa.

Fig. 2A. Lipiodol filling left nasal fossa, demonstrating occlusion of left posterior choana.

The X-ray examination of the sinuses (see Fig. 1) was negative except for the thickening of the lining of the left maxillary sinus; all the sinuses were fully developed. The left antrum was clear on irrigation. Fig. 2 illustrates the filling of the left nasal fossa with lipiodol and occlusion of the left posterior choana.

Powder blown into the nasal fossa on each side was promptly removed posteriorly by ciliary action. The ciliary movement appeared exceedingly fast on the obstructed side. A section of mucous membrane was removed from the anterior tip of the inferior turbinate for microscopic examination and was reported as follows (see Fig. 3): "The sections show

Fig. 3. Congenital choanal atresia. Photomicrograph tissue anterior portion of lower turbinate.

several collections of polymorphonuclear leucocytes in the fibrous tissue beneath the mucous membrane and a sprinkling of acute and chronic inflammatory cells throughout the fibrous tissue. The tissue is lined by normal appearing pseudostratified, ciliated, columnar epithelium. Aside from the acute and chronic inflammatory cells, the appearance is essentially that of normal nasal mucosa."

The tympanic membranes were dull and slightly retracted. The audiogram showed a bilateral symmetrical perceptive type of hearing impairment (see Fig. 4). The nasopharynx, except

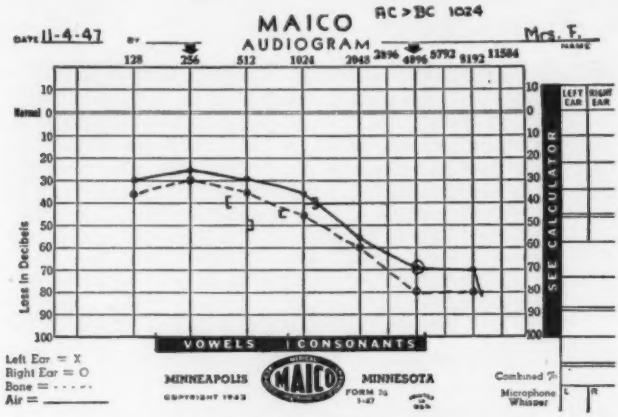

Fig. 4. Audiogram, Case 1.

for the occlusion of the posterior nares on the left side, did not appear unusual.

The stained smear of nasal mucus showed many polymorphonuclear leucocytes but no eosinophiles.

The second patient, age 19, had never breathed through her nose. She had complete anosmia. Except for wiping her nose frequently, this disability has never given her any particular trouble. She thinks she has more "head colds" than the average person. Her hearing has been adequate and her ears have never bothered her. As a baby, she heard her mother say she

was frail and had some difficulty in feeding; however, as long as she remembers she has never been sick. She is now the mother of a healthy baby girl. There was no family history of choanal atresia.

Examination showed a slender, attractive young lady. There was no facial asymmetry; the teeth were normal, and no palatal deformity was present. The tonsils were atrophic; the nasopharynx appeared normal except for the complete occlusion of each choana. The nasal mucous membranes appeared boggy with a bluish cast. The lower and middle turbinates were slightly atrophic and because of widening and lowering

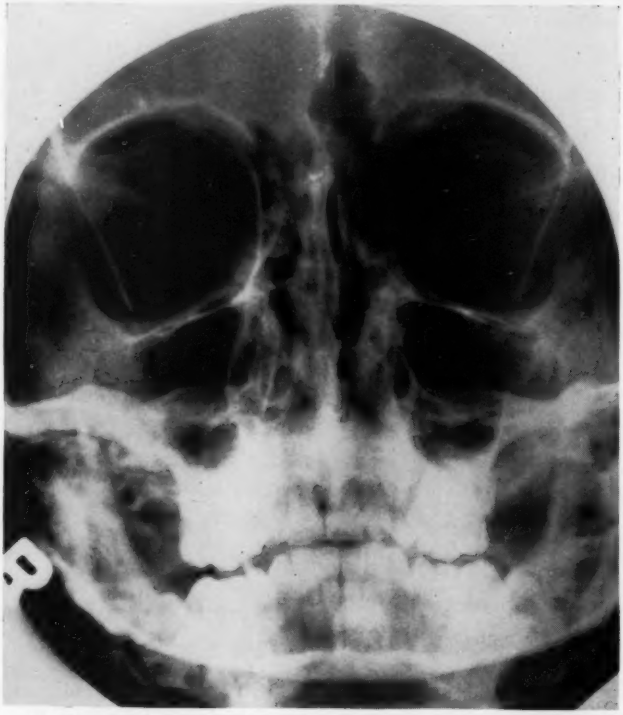

Fig. 5. Bilateral congenital choanal atresia. X-ray of sinuses.

of each nasal fossa and inferior meatus, appeared highly placed. The recessed nasal floors were filled with thin, glairy mucus. The inferior edge of each lower turbinate presented a slight mulberry degeneration. The posterior end of each fossa was completely occluded by a solid bony partition.

X-ray of the sinuses (see Fig. 5) showed small frontals especially on the right side. The maxillary, ethmoid and sphenoid sinuses were well developed; all of the sinuses were clear. Fig. 6 illustrates each nasal fossa filled with lipiodol.

Fig. 6A. Bilateral congenital choanal atresia. Lipiodol study.

The tympanic membranes appeared slightly retracted; audiometric examination showed normal hearing (see Fig. 7).

Fig. 6B. Bilateral congenital choanal atresia. Lipiodol in each nasal fossa.

Powder placed in each nasal fossa was rapidly carried posteriorly by active ciliary action. There appeared to be a triangular area of stasis at the anterior tip and along the inferior edge of each lower turbinate.

The only associated abnormality was a webbing of the second and third toes of each foot. General physical and laboratory examination was negative.

COMMENT.

In the majority of articles on congenital occlusion of the posterior nares the anatomic lesion and operative correction is

Fig. 6C. Bilateral congenital choanal atresia. Lateral X-ray with lipiodol.

described, but little mention is made of the anatomic development or disease of the sinuses. It can be assumed that these structures were normal in most instances. In the past it has been stated that all cases of choanal occlusion would sooner or later develop sinusitis, although the normal development of the sinuses was not impaired. Proetz[6] said, "It is generally agreed that stagnation of mucus in the closed side is apt to result ultimately in sinus disease." Grove[7] stated that, due to lack of air currents and free ventilation plus impaired ciliary action, the sinuses on the affected side were

prone to develop sinusitis. Dean[21] found all children whom he had examined with choanal atresia had sinusitis.

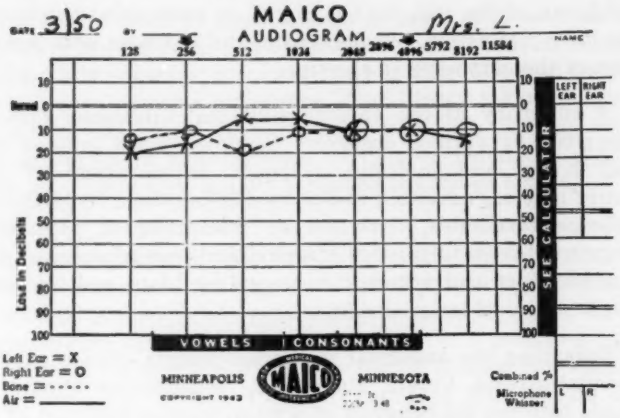

Fig. 7. Audiogram, Case 6.

A review of the cases of choanal atresia reported more in detail in the literature indicates that the anatomic development of the sinuses, in either bilateral or unilateral atresia, is unimpaired, and that sinusitis is an occasional rather than a routine finding. In 47 cases reported by 25 observers, 46 showed clinical or X-ray evidence of normal development of the paranasal sinuses. In one case the frontal sinus on the occluded side was underdeveloped. Four of the 47 cases showed evidence of sinusitis.

A similar analysis of the cases of congenital atresia reported in the literature indicates that aural involvement is not frequent. It is usually reported that the drums are thickened, dull or retracted, but the hearing is unaffected; in all but 10 of the 47 cases the hearing was essentially normal. Chronic suppurative otitis media was recorded in six cases. In one case the hearing was improved following correction of the atresia.

Regarding associated abnormalities, 10 of the 47 cases showed high arching of the palate, five cases showed asymmetry of the face, and one each showed bifid uvula, congenital coloboma of the iris, double tragus on each side, double congenital aural fistula, polydactylism, and flat nose with separation of the cartilages in the tip.

A summary of the various abnormal intranasal findings described by various observers includes: nasal mucosa pale and boggy and inelastic, polypoid degeneration of the entire mucosa, advanced mulberry degeneration, hypertrophic inferior turbinates, deviation or thickening of septum to obstructed side, turbinates atrophic and underdeveloped, turbinates short and attached to occluding plate and mulberry hypertrophy of lower turbinate.

Regarding the abnormal intranasal lesions associated with choanal atresia, Proetz[6] states that "a difference apparently exists between the behavior of a nasal mucosa deprived of air after breathing is established, and one in which, through congenital posterior closure, no breathing has ever taken place. In the latter condition normal, healthy tissues are often found in the nasal fossa, and there is also an absence of the hyperphasias and similar reactions usually encountered about the orifices of the Eustachian tubes in cases of acquired obstruction." Thomson[8] remarks, "It is noteworthy that this complete atresia of the nose, even with entire mouth breathing, does not entail the chest deformity, narrow nostrils, alar collapse, deflection of the septum, and changes in the ears which are frequently attributable to partial stenosis, or to chronic nasal infection, or to both." The most significant feature, therefore, is the difference between complete and partial nasal obstruction.

Except for the observation by Shambaugh[9] no mention has been found of the effect of choanal occlusion on ciliary function. Shambaugh studied the ciliary action after operation and reported normal function. In the cases here reported powder blown into each nasal fossa was quickly swept backwards, equally well on obstructed and unobstructed side.

Similarly, the sense of olfaction may be unaffected by choanal atresia; after surgical correction of the obstruction the sense of smell was restored in four of the 47 cases.

The few biopsies of nasal mucosa in cases of choanal occlusion have been reported in the literature and these have been significantly negative; generally there is little deviation for the normal nasal mucous membrane. The biopsy taken, as mentioned in Case 1, is in agreement with this finding, with the exception of the presence of ciliated columnar epithelium at a site where usually stratified squamous epithelium is found.

It is interesting at this time to compare the condition of the nasal mucosa in the laryngectomized patient and that seen in choanal occlusion. As reported by Dixon,[24] the color of the nasal mucosa after laryngectomy turns from a pink to a light purple, similar to the color of the allergic mucosa but without bogginess. The ciliary function appeared normal and the olfactory sense unimpaired, although the patients stated that the sense of smell was almost lost. The nasal mucosa was found uniformly moist; the dry, inactive, nonciliated areas became active. These patients did not have a postnasal discharge, and rarely had an upper respiratory infection. The microscopic examination of tissue removed from the anterior tip of the lower turbinate showed ciliated pseudostratified columnar epithelium in contrast to the stratified squamous epithelium seen in four out of five controls.

In Fig. 9 a summary of my six cases is made.

The treatment of congenital choanal occlusion is surgical. In the small child or infant the transpalatine approach[10] is advocated. Uusually the intranasal operation is used, with or without a submucous resection and removal of the posterior end of the nasal septum. If sufficient space is present the obstruction is adequately removed by direct approach, through the nares by means of perforating trocar and biting forceps and punches. Electrocoagulation has been reported by Morgenstern[11] as an effective simple office technique in adult cases, especially if the bone is thin. Wilkinson[2] and others advocated

the use of a long shanked dental burr. Successful approach to the atresia has been made in suitable cases via the transantral and trans-septal[12] route.

Fig. 9.

Case	Age	Type	Operation	Sinus, Ear or Associated Abnormalities
1	52	Unilateral—left	Refused	Bilateral nerve deafness not attributed to choanal atresia
2	20	Unilateral—right	Successful transnasal	None
3	45	Unilateral—right	Refused	None
4	15	Unilateral—left	Successful transnasal	None
5	Newborn	Unilateral—left	Deferred	None
6	19	Bilateral	Successful transnasal	Syndactylism

Many ingenious methods have been devised to maintain the patency of the choana postoperatively. Rubber and metal tubes have been found irritating and often lead to stenosis rather than prevent it. Gauze packing has many disadvantages. The most successful obturator is one made of acrylic.

SUMMARY.

The development and physiology of the nose and paranasal sinuses is apparently little affected by total unilateral and bilateral choanal occlusion. Many reports have been made and X-ray studies have been exhibited to illustrate complete development of the sinuses in cases of choanal atresia. Biopsy studies of the nasal mucosa have shown in the cases here reported, and in other recorded cases, no departure from the normal structure. The physiological activity of the nasal cilia appear to be unaltered by the presence of this congenital anomaly. These findings suggest, as Boies has stated in the discussion of Wilkinson's[2] paper, "that nasal respiration is not important to the development of the sinuses" nor, it would seem, to the normal physiology of the nose.

Sinusitis is an uncommon rather than a common complication of congenital choanal atresia. These patients are not prone to acute infections of the nose and sinuses; the incidence of acute rhinitis is less than average. The history of continuous "colds" is a result of the periodic drainage of the retained secretions.

The finding of aural disease and hearing impairment associated with choanal atresia is not common. Middle ear disease, if present, is often found on the unobstructed side. The audiogram is usually reported as normal. The tympanic membranes are usually described as dull, lusterless, thickened or retracted. The interesting audiogram in Case 1 shows a bilateral perceptive type of impaired hearing. Fig. 8[8] illustrates an essen-

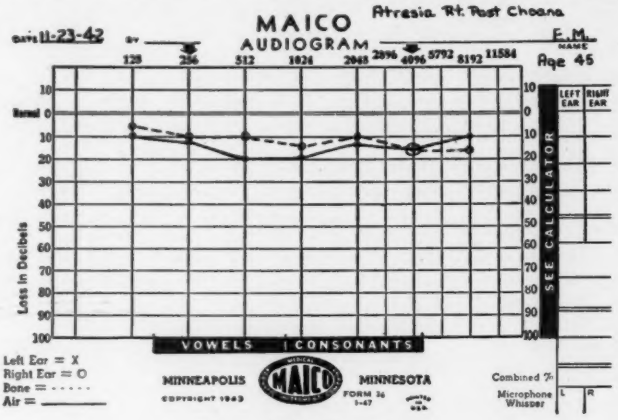

Fig. 8. Audiogram, Case 3.

tially normal audiogram of a 45-year-old colored woman (Case 3) with total right posterior nares occlusion. Wilkinson[2] reported improvement in hearing in one case after surgical correction of the occlusion.

Adult patients with unilateral or bilateral choanal occlusion, as a rule, complain of little discomfort or distress because of their disability; surgical correction is often refused. The accumulation of the thick mucus is removed by the patient by

gravity, the patient lowering the head and permitting the retained material to drain, assisted by pressure on the ala of the nose.

Bilateral choanal atresia in infants is of grave importance and requires prompt surgical correction; failure to recognize this condition has resulted in the death of infants within the first week of life, under the mistaken diagnosis of asphyxia neonatorium. Thompson[8] states that "the nose is the natural and instinctive channel for carrying on respiration. With rare exceptions all infants breathe entirely through the nose. If the nose of a sleeping child is gently closed, he will continue to make increasingly violent efforts to draw through it; when this proves useless, he will waken and gasp for breath, instead of simply opening and using the mouth as an airway. Even when mouth breathing is partially adopted to supplement the nasal airway, the latter remains the route preferred by instinct, and as much air as possible is drawn through it. Mouth breathing is an acquired habit." The diagnosis is suspected if the infant is unable to nurse, or has spells of suffocation and cyanosis which are relieved by crying and aggravated when nursing or sleeping. If the mouth breathing habit is established, the infant usually proceeds to normal growth and development except for the nasal obstruction and associated abnormalities. Unilateral atresia in the infant is largely a feeding problem and the prevention of malnutrition. Surgical correction may be undertaken in later life.

CONCLUSION.

Congenital choanal atresia is an unusual but not rare rhinological anomaly. The finding of the same defect in a mother and daughter suggests a definite hereditary tendency. Complete bilateral or unilateral atresia has surprisingly little effect on the development of the paranasal sinuses or on the physiology of the nose or ears. The association of sinusitis and choanal occlusion is uncommon. Aural disease and impaired hearing is infrequently seen in choanal occlusion.

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BILATERAL ABDUCTOR PARALYSIS. THE POSTOPERATIVE CARE AND SPEECH THERAPY FOLLOWING ARYTENOIDECTOMY.*†

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The postoperative care of bilateral abductor paralysis may be divided into surgical or medical problems and the rehabilitation of the patient's voice.

First let us consider the immediate care following surgery.[1,2,3] A drain has been placed and held in position by a skin suture. A snug pressure type dressing has been applied. Special nursing should be provided the first 24 hours to insure frequent suctioning of the tracheotomy tube. Coughing should be discouraged and no attempt made on the part of the patient to talk or whisper the first week. In those cases which have not had the benefit of a previously placed tracheotomy tube, sedation against coughing is generally needed. These are the cases in which coughing and undue strain might help to dislodge the lateralized cord. Unless there is some economic or other very good reason it is better to perform a two-stage procedure and have the tracheotomy tube placed before doing the arytenoidectomy. Fortunately, most of the cases come to us with the tube already installed. Such patients can be much more satisfactorily prepared medically as to their thyroid and parathyroid balance if the tube is in place, relieving them of their dyspnea. A recent article by Murtagh[4] has graphically demonstrated the results of improvement in ventilation and the return to normal of the red cell indices following relief of the marked dyspnea characteristic of these cases.

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If fluids are not taken readily by mouth the first postoperative day they are supported with infusions. It is rare that this is needed after the first 48 hours. We have not needed the aid of a feeding tube, though there is no reason why it cannot be used. Soft diet is generally tolerated after the third day.

All patients are under antibiotic therapy. We have used either sulfadiazine or crystacillin. This is continued until the wound is completely healed. It is not unusual to have a temperature of 101° the first postoperative day, which generally subsides to normal thereafter.

The drain is half pulled in 48 hours and completely removed on the third postoperative day. The stitches are half removed on the fifth day and all out on the seventh postoperative day.

The patient is urged to get out of bed the day following operation for a short period and in gradual amounts thereafter. Most of them are up and around after the third day.

No attempt is made to inspect the larynx with mirror until the third or fourth day. Little will be seen of either cord until the end of the first week. The amount of edema may then be checked regularly with the mirror and when the edema has subsided sufficiently and the glottic chink is adequate, tracheotomy tube corking is started. At first we use a half cork and work up to full cork as tolerated. They are decannulated after four days of full corking with trials of vigorous exercise and the patient feels well satisfied that he or she may carry on without the tube. Our average time for decannulating is 26 days. There is no advantage in rushing this until well satisfied with the airway obtained.

MEDICAL CARE – POSTOPERATIVE.

The postoperative medical care is made much easier if we have had preoperative determinations of the blood serum calcium, phosphorus and serum cholesterol. Holinger,[5] in 1944, was the first to emphasize this need in cases undergoing arytenoid surgery. The presence of a tracheotomy tube precludes the value of a basal metabolism test.

Thyroid activity may then be ascertained by radio tracer uptake or by serum precipitable iodine levels. The tracer uptake technique, however, may be radically modified by any recent ingestion of thyroid therapy as pointed out by Werner, Quinby and Schmidt,[6] which factor may limit its use, as many of these cases have been carried along on some thyroid medication.

One should be on the alert for those patients with low thyroid activity and parathyroid deficiency. The parathyroid lack in our series of cases has given the most alarming group of symptoms such as mental depression, anxiety, carpopedal spasm, and sometimes complete blackouts with tetanic convulsions. The latter group of symptoms are at times difficult to differentiate in a patient with a hysteria even though the blood calcium levels remain within normal limits. The patient, through hysteria and fainting spells with accompanying hyperventilation may help to bring on symptoms which are difficult to distinguish between parathyroid lack and hysteria. It is well to institute the parathyroid and thyroid therapy in any case of doubt. Those cases which appeared borderline have generally responded to such therapy even though the blood chemistry tests did not indicate the need of therapy. This suggests the possibility that not infrequently our means of measuring this deficiency is not always accurate and the patient should sometimes be treated empirically. This phase of the medical care should be under the guidance of an internist who is familiar with this problem.

In a few cases we have found an accompanying allergy responsible for some dyspnea, which is checked when the allergy has been corrected and controlled.

Those cases with parathyroid deficiency are the ones in greatest need of psychic therapy. Having them talk with previously operated patients is very helpful, and gives them reassurance. In these cases the patient should never be forced to go without the tube until he is well satisfied that he can get along without it.

SPEECH THERAPY.

While arytenoidectomy and lateralization of the vocal cord is primarily done for the purpose of giving the patients sufficient airway in order that they may carry on all of their normal activities without the use of a tracheotomy tube, we are also faced with the problem of rehabilitation of the voice.

When all evidence of postoperative edema has passed and the patient is decannulated, the patient is then started on speech therapy. They have already been seen by the speech therapist, who has told them what to expect in the line of training. Their voice has been recorded preoperatively and they have usually been introduced to other patients who have gone through the same training regime. This puts them at ease and helps to overcome the first hurdle they must meet — learning to relax. Inasmuch as most of these cases are among men or women who have had thyroid problems, many of them of the toxic variety, it is usual to find them apprehensive, worried, and totally lacking in confidence after their long experience of fighting for air and trying to overcome a voice handicapped by their dyspnea.

Postoperative patients find themselves relieved of air hunger but they now find their voices changed, not as the result of gasping for air but by the operated cord, which, if properly lateralized, is now fixed. This means an additional amount of adduction has to be accomplished by the unoperated side in order to get complete approximation for proper phonation.

Following training in relaxation, correct breathing is mastered. This is done through exercises and finally the third phase, that of timing the phonation to occur during expiration, is taught. Once these three fundamentals are accomplished, then various exercises for training in pitch are developed. With improvement comes added confidence and greater ease in building up the accessory mechanism which develop a good functioning voice.

It is true that a goodly percentage of these cases will have very satisfactory voices. Not singing voices, but voices which do not fatigue and can carry on a rapid and efficient conversation. Such voices are found among those cases where there has been the least amount of lateralization. This minimal amount may be well tolerated and satisfactory in a lightweight woman who does not have to do any physical work, but for those cases which need the extra airway resulting from greater physical demands, it is imperative that they have their cords lateralized to a 5 mm. glottic chink at least. These cases are particularly helped by speech therapy. There is also a group of cases, fortunately small in number, in which there has been too much lateralization, where the glottic chink is 6 mm. or more in width. Although this latter group has a voice adequate for all the functions of living and can even be understood over the telephone, their voices are distinctly rough and they, too, can be helped by speech therapy.

In a recent article, Clerf[7] made the observation and statement that "the wider the glottis is, the poorer the voice will be." Even though lateralization of the vocal cord may be carefully checked with the laryngoscope at operation, such factors as size, shape and rigidity of the laryngeal cartilages, together with edema and scar tissue secondary to operation enter into the resultant lateralization. While experience does help, the precise amount of lateralization is not exactly predictable.

Voice therapy for cases of aphonia and dysphonia postarytenoidectomy, as reported by Doreen Pollack, voice therapist of the Vanderbilt Clinic. Among patients attending the Vanderbilt Clinic, the following *symptoms* have been observed:

1. Anxiety with accompanying nervous muscular tension, because they are unable to produce a normal voice.

2. Harmful attempts to produce a voice, ranging from attempts to speak during inhalation, to harsh forced whispering.

3. Respiratory distress, with noisy shallow mouth breathing during speech, which results in an excessively breathy

phonation and prevents the voice from being audible or sustained, and may cause a feeling of pain in the chest and throat. This type of dysphonia was described by Jackson and Jackson[8] in 1935. This group of symptoms sets up a vicious circle, because the patient is embarrassed by the harsh whisper, and attempts to make this louder by using more muscular tension, which in its turn, leads to more respiratory distress. The noisy mouth breathing is, of course, a habit formed before the operation.

4. In most cases there is insufficient closure of the glottis (postoperatively) for a normal vocalization. If a vibration of the vocal cords can be produced, it is usually gruff and weak, lacking inflection.

The *voice therapy* used is based on normal voice production. This is explained to the patient in simple terms.

1. A steady expiration of breath through the relaxation of the chest walls and diaphragm.

2. Initiation of the vocal attack with expiration and without excessive muscular tension.

3. Use of the resonating chambers of face to give carrying power and tone to the voice, thus relieving laryngeal hypertension.

4. Use of the optimum pitch.

Aims of the therapy; and a brief summary of the techniques used:

1. Strengthening and regulating the excitation of the vocal cords, or in some cases, the ventricular bands, by teaching inaudible inhalation, and a slow, steady economical exhalation, through a conscious control over the movements of lower ribs and diaphragm. It is important for the patient to keep the upper chest and shoulders firm. This control is then used in simple speaking exercises; counting, repeating phrases, and reading aloud.

2. The laryngeal muscles are strengthened and the glottis closed by various "pushing" exercises, following the principle that pressure exerted by hands and arms with a firmly held chest tends to close the glottis. (For example: the closed fists are held at the level of the nipples, and the patient pushes sharply downwards to the thighs, simultaneously saying AH or OO, and then other vowels. Pushing is then performed with voiced consonants and the vowels; single words; then phrases, and finally short sentences with only one push to initiate the sentence.) The therapist can also assist the action of the cords by finger pressure on the thyroid cartilage. Swallowing, followed immediately by vocalizing on EE, is sometimes helpful.

3. Resonance (*i.e.*, humming exercises) on nasal consonants, and the words containing nasal consonants, with the sensation that the voice is being sent into the front of the face is developed to produce a full tone, and relaxation of tension in the neck.

4. Finally, pitch exercises, including intoning on different notes, are given to increase the flexibility of the voice and to find the optimum pitch.

LENGTH OF TREATMENT.

This varies, but the following may be helpful:

1. Treatment must be regular, and at first should be given at least twice a week so that the therapist can be certain that the exercises are being practiced correctly.

2. Breathing, pushing and resonance exercises show marked results after one or two months, and the patient can then concentrate on finding the optimum pitch.

3. Some of the initial difficulties are caused by failure to keep the upper chest and shoulders firm, and to breathe deeply.

4. Success of the therapy depends upon the persistent practice done by the patient. Practice periods will be short at first, lengthening to about 20 minutes, but they must

be frequent. Should the patient neglect the practice during the first few months, a relapse will occur immediately. With regular and frequent practice periods, and real attempts to apply the practice during conversation, vocalization becomes effortless, and increased feeling of self-confidence and good muscular tone is noticeable.

5. It is worth mentioning that some patients experience greater difficulty during damp weather, and may relapse if harassed by family problems.

SUMMARY.

We have reviewed the postoperative care of cases operated upon for bilateral abductor paralysis and we have included the surgical and medical needs of these patients together with the value of speech therapy in voice rehabilitation.

A Kodachrome Sound Track Film followed demonstrating the open approach to arytenoidectomy with lateralization of the vocal cord. Views of vocal cords and voice recordings both before and after surgery were shown. This technique has been carried out by many surgeons thoughout this country, Europe and Canada. A total of over 150 cases have been reported to the author. The film emphasizes the importance of the role played by speech therapy in the rehabilitation of these cases.

Copies of this film may be obtained or rented through Sturgis-Grant Productions, 314 East 46th Street, New York 17, N. Y.

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CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The next meeting of the Central Illinois Society of Ophthalmology and Otolaryngology will be held at the Abraham Lincoln Hotel in Springfield, Ill., on Sept. 22, 23 and 24. The guest speakers will be Dr. Girolamo Bonaccolto, of New York, who will give several lectures on ophthalmology, and Dr. Bayard T. Horton, of the Mayo Clinic, who will speak on "Histaminic Cephalgia and the Treatment of Retrobulbar Neuritis with Histamine." Dr. C. L. Pannabecker and Dr. A. L. Paisley, two of the members of the Society, will also speak.

In Memoriam

GEORGE W. WALLERICH,

1880-1950.

George W. Wallerich, president of V. Mueller & Co., Chicago, Ill., died July 3 after a long illness. Mr. Wallerich, who was born on April 3, 1880, had been an eminent leader in the surgical instrument industry for more than 45 years. Forty years of this time were spent with the Chicago surgical instrument house which he joined as secretary shortly after his marriage to Vinzenz Mueller's daughter, Marie. Previously he was sales manager of the Victor X-ray Corp., during which time he was regarded as one of the country's outstanding authorities on X-ray equipment and techniques.

With his father-in-law, Mr. Wallerich threw his energy and imagination into the problems of the then infant American Surgical Trade Association. Together they founded and co-authored the *ASTA Journal* at their home at 2827 Jackson Boulevard, which served as the magazine's first offices. After business hours, Vinzenz Mueller edited the copy and George Wallerich tried to solicit enough advertising to keep the budding journal going.

As a lieutenant colonel, later a colonel, Mr. Wallerich served with the Sanitary Corps, U. S. Army, in World War I. During World War II, he was a member of the National Research Council.

In professional circles, Mr. Wallerich's name was respected throughout the world by medical men who sought his aid in the development of new surgical instruments and equipment. Much of his time was spent in working out improvements in old instruments for surgery and in developing new instruments for new techniques. Mr. Wallerich's intense interest in his work carried over into his personal life, and many of his closest friends were surgeons and hospital executives. He was, according to one of the latter, "a man of very high intellect and broader vision than the average man, a conscientious worker. . . ."

Mr. Wallerich became president of V. Mueller & Co. upon the retirement of Vinzenz Mueller. He was a member of the Presbyterian Church, the Oak Park Country Club and the Chicago Athletic Club.

Mr. Wallerich is survived by his wife, Marie Mueller Wallerich; a son, George Mueller Wallerich, vice-president of the firm; a daughter, Mrs. Constance Bruckner; and four grandchildren.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of April 1, 1950.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer; Maico UE-Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; tarion Model E-4; Otarion Models F-1 and F-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 103BM.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.*, 109:585 (Aug. 21), 1937.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.*, 139:785-786 (Mar. 19), 1949.
Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.*, 141:658 (Nov. 15), 1949.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Philip E. Meltzer, 20 Charlesgate, West Boston 15, Mass.
Secretary: Dr. Gordon D. Hoople, Medical Arts Bldg., Syracuse 3, N. Y.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Green Briar Hotel, White Sulphur Springs, W. Va., May 9-10, 1951.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

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Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October 3-6, 1950.
Hotel Biltmore, New York, N. Y., January 8-11, 1951.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles, Calif.
President-Elect: Dr. Derrick Vail, Chicago, Ill.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October 8-14, 1950.

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Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Secretary-Treasurer: Dr. Victor Goodhill.
Chairman of Section on Ophthalmology: Dr. Dennis V. Smith.
Secretary of Section on Ophthalmology: Dr. Carroll McCoy.
Chairman of Section on Otolaryngology: Dr. Howard P. House.
Secretary of Section on Otolaryngology: Dr. Edwin Scobee.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Alfred Schattner, 115 E. 61st Street, New York 21, N. Y.
Secretary: Dr. Norman N. Smith, 291 Whitney Avenue, New Haven 11, Conn.

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President: Dr. G. M. Billings, Morganton, N. C.
Secretary and Treasurer: Dr. MacLean B. Leath, High Point, N. C.
Time and Place: Sept. 11-13, Hendersonville, N. C. Jointly with the South Carolina Society of Ophthalmology and Otolaryngology.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. David R. Higbee, 3245 4th St., San Diego, Calif.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco, Calif.

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Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: St. Louis, Mo., November 13-16, 1950.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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